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DEPARTMENT OF REVIEWS

The Journal will make an especial feature of the review of monographs and books bearing upon the field of Internal Medicine. Authors and publishers wishing to subject such material for the purposes of review should send it to the editor. While obviously impossible to make extended reviews of all material, an acknowledgment of all matter sent will be made in the department of reviews.

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Epilepsia Tarda*

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GENERALIZED convulsions occurring for the first time in adult life are not infrequently viewed as diagnostic of uremia or of some gross brain lesion. Such preconception, however, leads to many errors in diagnosis and prognosis.

It is our purpose to call attention anew to the recently somewhat neglected subject of so-called epilepsia tarda and to attempt an analysis based on our own experience and a review of the literature.

Great confusion exists concerning the meaning of the term epilepsia tarda (senile epilepsy). Some authors (1) (2) (3) would restrict it to those cases of "idiopathic epilepsy" of later life which present no evidence of cerebral arteriosclerosis or other organic disease. Others on the contrary—and this includes most of the modern writers—feel that senile epilepsy is in some way related to disturbances of the cerebral circulation incident to cerebral arteriosclerosis and atheroma. In surgical literature one finds "epilepsia tarda" designating cases of delayed Jacksonian convulsions due to prior cranial injury. Further confusion is obvious from the statement of a widely used medical dictionary that "senile epilepsy is senile bradycardia coming on in paroxysms and resulting from coronary sclerosis....".

We would label with the term epilepsia tarda those patients who exhibit recurrent generalized epileptiform seizures after forty years of age and who fail to show any sufficient etiologic factor other than a background of circulatory inadequacy, with special reference to periodic insufficiency of the cerebral circulation.

This tentative definition commits us to the hypothesis that the convulsions of epilepsia tarda are initiated, in part at least, by moments of deranged cerebral circulation—the pre-existing background for which we believe is usually demonstrable. That there is another factor—the unknown X of all the forms of epilepsy must be admitted. An approach to this unknown, we feel, may be made possible through a study of the types of epilepsia tarda in which a knowable factor seems to operate.

Patients with epilepsia tarda as observed by us may be conveniently divided into groups as follows:

1. The arteriosclerotic group.
2. The hypertensive group.
3. The mixed hypertensive-arteriosclerotic group.

*Read before the Association of American Physicians, Atlantic City, N. J. May 2 and 3, 1927.

4. The endocrine dysfunction group, including (a) diabetes mellitus, (b) dyspituitary and hypothyroid disorders.

5. The cardiac group, including (a) Adams-Stokes disease, (b) aortic stenosis, etc.

No cases of group five are included in the present report. Nor is any mention made of those allied conditions of transient paralysis and aphasia described by Osler (4), Riesman (5) and others. The convulsions of general paresis are also omitted in our report—although they too doubtless belong to the arteriosclerotic group of epilepsia tarda. As may be seen from the following case reports the groups are by no means sharply defined, but merge one into another.

CASE REPORTS

Case No. I. (Arterosclerotic Type) Mrs. F. G.—Age 69. 11/6/25—Patient was well until two years ago when she fell unconscious on stairs and was found in a generalized convulsion. No cranial injury but arm was broken. Convulsion lasted several minutes; unconsciousness lasted an hour. Since then the patient has had ten attacks of unconsciousness with epileptiform seizures —three diurnal and seven nocturnal. No aura. She is dazed after consciousness returns and speech is thick but no residual paralyses have occurred. Recently she had two convulsions in one night. Incontinence of bladder and bowel in several attacks, but no tongue biting. Patient has had in addition two attacks of momentary unconsciousness without convulsions. Disposition becoming irritable; memory impaired; outbursts of causeless laughter recently. Some vertigo at times preceding the attacks and also without relation to attacks. Constipation always. Never had juvenile convulsions.

Examination: P.88; B. P. 150/60; W. 173. Ophthalmoscopic examination reveals marked retinal angiosclerosis. Heart enlarged to left about 3 cm. and downward to 6th interspace in anterior axillary line. Occasional extrasystyoles. Neurologic examination negative. Peripheral arteries hard. Urine: acid, 1012, no sugar, a trace of albumin, many pus cells, a few hyaline casts.

Summary diagnosis: (1) Epilepsia tarda (2) Arteriosclerosis (3) Cardiac hypertrophy with extrasystoles.

Case No. II. (Arteriosclerotic Type) Mr. W. H., negro—Age 55. 11/12/23—For the past year but never before patient has had about twelve generalized convulsions with unconsciousness and tongue biting and urinary incontinence. Attacks chiefly nocturnal. No aura. Nothing else of significance in history except occasional nocturia and bleeding hemorrhoids.

Examination: P.60 to 70; B.P. 120/70. Well developed negro. Marked retinal angiosclerosis but no demonstrable hardening of the peripheral arteries. Heart negative except for a blowing systolic murmur at the mitral area. Neurologic examination negative. Urinalysis negative. Blood and spinal fluid and Wasserman negative. Blood urea nitrogen, blood uric acid, blood sugar—all normal. Blood count moderate secondary anemia. Electrocardiogram shows sinus bradycardia.

Summary Diagnosis: (1) Epilepsia tarda. (2) Cerebral arteriosclerosis. (3) Secondary anemia from hemorrhoids. (4) Systolic cardiac murmur.

Case No. III. (Arteriosclerotic Group) —Mrs. R. P.—Age 74. 4/20/27—For the past year patient has had nocturnal convulsions every few weeks with stertorous breathing, swallowing movements and bladder incontinence. False teeth are removed at night and tongue is not injured by the biting which occurs. No aura. Some mental irritability. Constipation for years. No juvenile convulsions. No epilepsy in family.

Examination: Wt. 138; P. 92; B. P. 164/90. Peripheral arteries hard. Well marked ret-

inal angiosclerosis. Thyroidal adenoma. Heart enlarged with left border one inch beyond mid-clavicular line. No murmur. No arrhythmia. Urine: acid, 1015, albumin a trace, no sugar, a few hyaline and light granular casts.

Summary Diagnosis: (1) Epilepsia tarda. (2) Arteriosclerosis. (3) Cardiac hypertrophy.

Case No. IV. (Hypertensive Type)—Mrs. D. C.—Age 48. 11/24/26—Patient has known that her blood pressure was too high for the past eight years. In 1918 began to have headaches, vertigo, tinnitus and shortly thereafter had her first convulsion. From 1918 to 1924 she had repeated attacks of unconsciousness with generalized convulsions lasting from fifteen to twenty minutes and followed by mental dullness. Aura of "something rising up from epigastrium." No tongue biting or incontinence. Attacks all diurnal. No petit mal. Has had no attacks for two years. No juvenile convulsions. Patient also complains of gaseous indigestion and crying spells and constipation. Menopause three years ago at forty-five.

Examination: Overweight (159); B. P. 240/140; P. 104. Heart enlarged 2 cm. to left of mid-clavicular line. Area of supracardiac dullness increased. Aortic second sound accentuated. Tenderness over the gall-bladder area. Reflexes normal. No demonstrable arteriosclerosis. Urine: acid, 1020, albumin trace, sugar negative, microscopy negative. Blood urea nitrogen and blood sugar normal. Phenolsulphonephthalein elimination 45% in two hours.

Summary Diagnosis: (1) Epilepsia tarda. (2) Hypertension. (3) Gall bladder disease. (4) Cardiac enlargement. *Course*: Patient has been symptomatically improved by low salt low protein diet together with biliary drainage, acidophilus milk, and the use of bromides and luminal and colonic irrigations. Last examined 1/17/27—Wt. 157; B. P. 212/118. No more convulsions.

Case No. V. (Mixed Arteriosclerotic Hypertensive type)—Mr. E. H. Age 59. 9/15/25—For five years has had dyspnea on exertion and edema of the feet. In September 1923 had his first convulsion and had recurrent convulsions with unconsciousness about once a month for the next year—always nocturnal and associated with tongue biting and bladder incontinence at times. Previous history includes gonorrhea and inflammatory rheumatism. No convulsions have occurred during the past year since his doctor (R. D. Anderson of Burlington, N. J.) began the use of luminal.

Examination: Overweight; P. 80; B. P. 180/100. Marked peripheral arteriosclerosis and retinal arteriosclerosis as well. Marked cardiac hypertrophy with engorged liver extending three inches below costal border and a blowing systolic mitral murmur. Some edema of legs and feet. Prostate markedly enlarged and urethral stricture reported by urologic examiner. Urine loaded with pus. Blood urea nitrogen 38—reduced to 18 by inlying catheter. Blood Wassermann negative.

Summary Diagnosis: (1) Epilepsia tarda, (2) Arteriosclerosis and hypertension, (3) Cardiac hypertrophy with decompensation, (4) Prostatism. *Course*: The constant use of luminal in half-grain doses seems to have completely abolished convulsive seizures. He died 3/31/27 of cardiac decompensation and uremic (?) coma without convulsions or paralyses.

Case No. VI. (Mixed Arteriosclerotic-Hypertensive type)—Mr. M. H. Age 56. 11/7/22—Patient has had attacks of vertigo for the past year with occasional headache and palpitation.

Examination: B. P. 196/100; P. 84; Wt. 196. Patches of leukoplakia inside cheeks. Faint systolic murmur at aortic area and slight left-sided cardiac enlargement. Moderate edema of shins. Peripheral arteries moderately sclerosed. Arcus senilis present. Urinalysis: 1028, albumin trace, sugar negative, microscopy negative. Blood Wassermann negative.

12/14/22—Fell down stairs in a "dizzy spell." No witness and no knowledge of convulsion. 1923—Had broncho-pneumonia. Blood pressure tending higher. 3/24/26—

While on ocean trip from Bermuda (3/14/26) had sudden vertigo and lapsed into unconsciousness. Eye witnesses state that he had a violent convulsion with bloody froth from mouth. Vomited on regaining consciousness.

Examination: B. P. 210/110; Wt. 184; P. 76. Urine as before except more casts. Blood urea nitrogen, uric acid, sugar, and creatinin normal. B. S. P. 35% elimination in two hours. Electrocardiogram shows "severe myocardial degeneration." Opthalmoscopic examination (Dr. Fewell) "marked angiosclerosis. No hemorrhages." 7/16/26—Generalized convulsion with tongue biting followed by visual hallucinations which lasted several weeks. 9/4/26—Convulsion preceded by aura of vertigo and precordial distress. Occasional extrasystoles noted after this attack. 3/7/27—Convulsion with severe laceration of the tongue. Blood pressure in the attack was over 300 mm., pulse was 120 and gallop rhythm developed with a grating systolic murmur over the entire heart. Patient passed sixty ounces of urine in the three hours following this attack. No sphincteric incontinence at any time. 4/10/27—Convulsion as above. A good deal of anginoid discomfort.

Summary Diagnosis: (1) Epilepsia tarda. (2) Hypertension and arteriosclerosis. (3) Myocarditis.

Case No. VII. (Mixed Hypertensive—Arteriosclerotic-luetic). Mr. W. B. T. Age 58. 2/2/24—For the past year patient has had recurrent generalized convulsions with unconsciousness, tongue biting and urinary incontinence. Most of the attacks are nocturnal and are frequently preceded by aura of precordial distress. Has been overweight for many years. Was first told he had high blood pressure two years ago. Chancre at twenty-one years of age. No juvenile convulsions. No epilepsy in family.

Examination: Wt. 202; B. P. 215/115; P. 80. Large and obese. Eye grounds show marked arteriosclerosis and small patch of choroiditis inside both discs. Neurologic examination negative except absent left knee jerk. Heart enlarged with left border at anterior axillary line. Rough systolic murmur at aortic area transmitted to neck. Urine: 1020, no sugar, albumin a trace, a few granular casts. P. S. P. 50% in two hours. Blood Wassermann plus four.

Summary Diagnosis: (1) Epilepsia tarda. (2) Hypertension and arteriosclerosis. (3) Syphilitic aortitis and cardiac hypertrophy. *Course*: Patient improved under antisyphilitis treatment, dietary management and the use of luminal. Convulsions did not recur for full year when patient stopped treatment. Last heard from in March 1927 (aet 61), he had suffered a right hemiplegia.

Case No. VIII. (Endocrine Dysfunction type: Diabetes Insipidus). Mrs. R. S. Age 53. 2/22/27—Patient was well until menopause at forty-six when she had vertigo and flushes and has not felt well since. A year ago she was told for the first time that she had high blood pressure. During the past month she has had two attacks of unconsciousness with jerking of the head and face and limbs but no tongue biting or incontinence. Attacks preceded by aura of choking feeling in throat and burning feeling in epigastrium and followed by dazed mentality for several hours. No nocturnal attacks. No petit mal. Ever since first attack has had very marked polydypsia and polyuria—passing five to six quarts of urine in twenty-four hours. Has become very nervous and memory is failing. No juvenile convulsions. Weighed 150 pounds at marriage and went up to 208 pounds a year ago. Comes of fat stock.

Examination: Wt. 197; P. 92; B. P. 170/80; "Full moon face." Lower eye lids puffy. Tonsils very large. Heart negative except for a soft systolic murmur at apex in recumbent posture. Abdomen adipose. No demonstrable arteriosclerosis. Neurologic examination, negative except knee jerks greatly diminished. Urine 1006, no sugar, no albumin. Blood Wassermann and Kahn tests negative. Blood sugar: .98% (fasting).

Summary Diagnosis: (1) Epilepsia tarda. (2) Diabetes insipidus. (3) Hypertension. (4) Heart murmur.

Case No. IX. (Endocrine Dysfunction type: Hypothyroidism. Arteriosclero-

sis). Mrs. J. M. Age 53. 12/20/21—Patient was well except for obesity and dyspnea on exertion until two months ago when she began to have convulsions of brief generalized type. Has had about fifteen such attacks in past two months. Following attacks she is confused and recently has been childish and forgetful. Has aura of precordial distress at times. In several of the attacks the tongue has been bitten slightly and the bladder has emptied incontinently. Always constipated.

Examination: B. P. 120/70; P. 100; Obese. Hair of scalp sparse and dry. Face and hands suggests myxedema. Opthalmoscopic examination (Dr. Baer) "marked retinal angiosclerosis with a few recent hemorrhages around both discs." Heart enlarged 3 cm to left of mid-clavicular line. Sinus arrhythmia and tachycardia at times and at other times slow rate. Sounds muffled. Large umbilical hernia. Neurologic examination negative. Repeated urinalyses, blood counts, blood chemical examinations, blood Wassermann—all normal. Basal metabolism (during afebrile period when pulse rate was averaging about sixty-two) was minus 16%.

Summary Diagnosis: (1) Epilepsia tarda. (2) Arteriosclerosis. (3) Cardiac hypertrophy. (4) Hypothyroidism. *Course*: Treatment by rest in bed, low salt low caloric diet, and colonic irrigations together with bromides and thyroid extract was followed by a period of eleven months of marked improvement. The convulsions ceased and the mental condition cleared up entirely. Thirteen months after onset the patient had hemiplegia and died.

Case No. X. (Endocrine Dysfunction type—Diabetes Mellitus with Hypertension and Arteriosclerosis). Mr. W. P. Age 50. 11/15/26—Well until four years ago when he had right renal colic. Three years ago right nephrectomy for nephrolithiasis. Felt well until May 1926, when he had a convulsion which left him with some motor and visual aphasia but no other paralysis. Second convulsion today, 11/15/26, generalized with tongue biting and followed by a deep sleep lasting two hours.

Examination: Wt. 170; B. P. 250/140; P. 60. Eye-grounds show silver wire arteries indenting the somewhat dilated veins. No cranial nerve palsies. Heart slightly enlarged. Rough systolic murmur at aortic area and marked accentuation of second aortic sound. Reflexes normal. No edema. Blood urea nitrogen—28 mgms. per 100 cc, blood sugar 199 mgms. per 100 cc. Urine loaded with pus and containing 1% of sugar and a heavy trace of albumin. Specific gravity 1020. Urinary tract: X-ray shows several small calculi left kidney. Blood Wassermann negative.

Summary Diagnosis: (1) Epilepsia tarda. (2) Hypertension and Cardiac hypertrophy and aortitis and arteriosclerosis. (3) Diabetes Mellitus. (4) Nephrolithiasis. *Course*: 4/4/27—Under diabetic management blood sugar is normal. Urine is free of sugar and no more convulsions have occurred but hypertension, aphasia and pyuria persist.

Case No. XI. (Endocrine Dysfunction type: Diabetes Mellitus, Arteriosclerosis). Mrs. L. McL. Age 59. 11/2/22—Diabetes was discovered at the age of seventeen. After one year of treatment patient considered herself cured and apparently remained well for the next forty years when loss of weight, polyuria and polydypsia recurred and sugar was again found in the urine (1920). Soon after this patient began to have attacks of unconsciousness with convulsions (aet 57).

Examination: B. P. 100/70; P. 72; Wt. 95. Considerable emaciation. Marked peripheral arteriosclerosis. Heart enlarged with left border an inch beyond mid-clavicular line. Marked edema of legs. Neurologic evidence of tabes diabetica. Urinalyses: variable amounts of sugar. Occasional ketones. Fasting blood sugar varies from .295 to .500%. Blood urea nitrogen 19 mgms. Wassermann negative.

Summary Diagnosis: (1) Epilepsia tarda. (2) Diabetes Mellitus. (3) Arteriosclerosis. (4) Cardiac hyperthrophy. *Course*: After the patient's regime of diet and insulin was properly regulated she had no more convulsions until 1926 when she broke diet. At present (4/7/27) she is quite well with urine free of sugar.

Case No. XII. (Endocrine Dysfunction type—Diabetes Mellitus with Hypertension and Arteriosclerosis). Mrs. D. M. Age 53. 10/3/23—Well until one year ago when pruritus vulvae and polyuria led to finding of sugar in urine. Had first convulsion nine months ago while cooking over hot stove. Second convulsion three weeks ago while ironing. Both preceded by vertigo and followed by heavy sleep for several hours. No tongue biting. No sphincteric incontinence. About ten days ago had a series of three brief convulsions without regaining consciousness between convulsions. No residual paralysis and no petit mal attacks but some loss of memory recently. Menopause six years ago. Two brothers died of diabetes. No epilepsy in family.

Examination: B. P. 195/120; Wt. 175; Height 65 inches; P. 72. Obese. Eyegrounds show marked indentation of veins by silver wire type of arteries. Heart shows occasional extrasystole. Left border is 3cm. to left of mid-clavicular line. Rough systolic murmur at aortic area. Urinalysis: Sugar 1.8%. No Ketones. Blood sugar, .245%. Blood urea nitrogen eighteen mgms. Wassermann negative.

Summary Diagnosis: (1) Epilepsia tarda. (2) Diabetes Mellitus. (3) Hypertension. (4) Cerebral Arteriosclerosis. (5) Cardiac Hypertrophy. *Course*: 4/15/27—Patient has been uncooperative as regards diet. Has had three convulsions in past two years. Following the last convulsion three months ago developed a right hemiplegia which has now almost disappeared.

Summary of the salient features of these twelve case-histories reveals some interesting facts. Five are men, seven are women. Nine are overweight. The average age at onset of convulsions is fifty-six years—with forty-one as the youngest and seventy-three as the oldest age of onset. Regarding the character of the seizures, all are of the recurrent, grand mal type. In two patients the attacks are exclusively nocturnal, in four exclusively diurnal. Two patients exhibit, in addition to major fits, petit mal attacks. An aura, usually of precordial distress or vertigo, is noted by six patients. One case-history reveals an episode of status epilepticus. Tongue biting is mentioned in five cases, sphincteric incontinence in six. The only classical feature lacking in these convulsions is the initial cry.

The grouping of our cases is as follows: three arteriosclerotic, one hypertensive, three mixed hypertensive-arteriosclerotic (one of which is syphilitic), and five belong to the endocrine dysfunction group. Of the latter, three are cases of diabetes mellitus, one of hypothyroidism, and one of diabetes insipidus. Every case in this endocrine dysfunction group has either hypertension or arteriosclerosis or both in addition. In no case is there any evidence of marked impairment of renal function—except in case five which terminated in the uremia of prostatism five years after the initial convulsion.

The only underlying condition present in all cases is some circulatory abnormality. These abnormalities are divided as follows: (1) All twelve patients exhibit some cardiac disturbance, ten with definite hypertrophy and varying degrees of myocardial degeneration and two without demonstrable hypertrophy but with well marked systolic apical murmurs (in addition one of the latter shows sinus bradycardia in the electrocardiogram). (2) Ten of the twelve present definite evidence of arteriosclerosis as judged both by palpation and by ophthalmoscopic examination. (3) Seven are hypertensive. (4) Eleven patients present two or more of the above factors combined.

Only one patient (case 8) has neither demonstrable cardiac hypertrophy nor demonstrable arteriosclerosis, but she has hypertension and diabetes insipidus.

Prognostic points in our series are as follows: Two of the twelve patients have died—case 5 in coma five years after initial convulsion and case 9 of apoplexy one year after onset. Eight patients are alive—averaging at present almost four years of life since onset of convulsions. Two patients have not been heard from. Of the eight known living patients, two have had strokes and are confirmed invalids, two others have such marked myocardial and hypertensive disease as to be totally incapacitated, while four are able to lead fairly normal lives. The longest duration of life after the initial convulsion is that of case 4 (of the hypertensive group). This patient is alive and well (except for a blood pressure of over 200) now nine years since her first fit and three years since her last one. The second best showing is made by one of the diabetic group (case 11) for whom insulin came just in time and who is now well seven years after convulsions began.

With the facts of these case histories in mind let us return to a brief discussion of our belief that moments of deranged cerebral circulation are responsible in part at least for the convulsions of epilepsia tarda. Of course this thesis in regard to epilepsy in general is not original with us. Sir William Gowers (6) has emphasized it. Russell in his Goulstonian Lecture in 1909 (7) maintained that "the fundamental factor underlying both the ordinary faint and the epileptic fit is cerebral anemia." Moon (8) presents a recent concise review of this hypothesis and reports a case of mitral stenosis with epilepsy at thirty-five. This report indicates the impossibility of sharply separating so-called idiopathic epilepsy from senile epilepsy.

It is well known that sudden disturbances of the cerebral circulation are sometimes followed by convulsions. They may occur, for example, during the rapid aspiration of pleural effusion, following profuse hemorrhage from any cause and they have been produced in man and animals by compression or ligation of the carotid arteries. In three patients with senile epilepsy Naunyn (9) was able, by manual compression of the carotids, to reproduce typical grand mal fits. He was also able to initiate fits in a few other non-epileptic but definitely arteriosclerotic individuals, whereas carotid compression failed to produce convulsions in a group of healthy adults. A recent significant and important study by Bordley and Baker (10) indicates that localized cerebral arteriosclerosis may be present without clinical evidence, and furthermore that such arteriosclerosis was found in all their cases of "essential hypertension." Just how the group of endocrine dysfunction cases of senile epilepsy might fall in line with our circulatory hypothesis is a little difficult to state. Of course all of our cases had, in addition to diabetes mellitus or insipidus or hypothyroidism, some definitely organic cardiac or vascular defect. We feel that the endocrine dysfunction factor is to be viewed chiefly as a cause predisposing to organic circulatory disease. The possibility, however, of sudden circulatory changes of

hormonal nature (comparable to the convulsions of insulin shock) cannot be excluded. We certainly do not agree with the prevailing French view (11) which would ascribe to acidosis the diabetic type of epilepsy. It is interesting to note in passing that the first allusion in American literature indicating a recognition of the diabetic type of senile epilepsy is that of Dr. Phillip S. Roy in a discussion of this subject in Washington, D. C., thirty years ago. (12).

Diagnosis of the several types of epilepsia tarda is by no means always easy. The physician who is called upon for an opinion either during or immediately following the initial convulsion certainly has many possibilities to consider. We may mention the more probable causes of generalized convulsions of adult life at this point: apoplexy, hysteria, general paresis, eclampsia, acute yellow atrophy, meningitis, encephalitis, brain tumor, Adams-Stokes disease, coronary artery occlusion, intoxication by alcohol, strychnin, belladonna, lead etc., sudden anemia from hemorrhage, cranial trauma, brain abscess, the various types of senile epilepsy, and finally uremia. It is the group which comprises the senile epilepsies, apoplexy, and uremia that offers the greatest difficulties to the diagnostician.

When more than one convulsion has occurred and an interval of comparatively normal health has intervened the diagnosis is easier. Such a history points strongly to one of the forms of epilepsia tarda, the diagnosis of uremia which is usually made is seldom correct. The difference in prognostic significance is very great—a patient with true uremic convulsions is as a rule at death's door, a patient with epilepsia tarda may live for many years and may indeed be cured of his convulsions.

It is not sufficiently appreciated that generalized convulsions are rare in true uremia. It is significant, we think, that Strauss (13) noted no increase in the incoagulable nitrogen of the blood in cases of so-called uremia with convulsions. Agnew (14) states that "convulsions are probably not a part of pure nitrogen retention." In a more recent study of the syndrome of azotemia Feinblatt (15) found uremic convulsions surprisingly infrequent and when they did occur they were always terminal—in fact within an hour of death. We do not feel that the very rare condition of uremia without azotemia (16) is deserving of mention in this discussion. It would almost seem that azotemia inhibits epileptiform seizures. It follows that the diagnosis of uremia in an elderly subject with an initial convulsion should not be made hastily. One of the types of epilepsia tarda must be borne in mind.

In the treatment of epilepsia tarda the associated and underlying pathologic processes must be regulated as far as possible. As in idiopathic epilepsy so here the emphasis must be placed upon a quiet even mode of life with all sources of irritation—both physical and mental—eliminated as far as possible. A bland diet of the low salt, low protein type and small meals seems to us to be the most suitable. Constipation must be rigidly combatted. Bromides or luminal are usually indicated. Iodides may be tried. Thyroid extract seems to

have benefitted the convulsions in our case of hypothyroidism. Other endocrine substances are recommended but we have not been able to convince ourselves of their efficacy—always excepting insulin for the diabetic patients who need it. Digitalis and diuretics are sometimes indicated. In emergencies venesection has seemed helpful, as in one of our hypertensive cases.

The prognosis like the treatment varies with the underlying conditions. The cerebral arteriosclerotic "epileptic" may live for many years, although progressive mental deterioration is the rule. Sometimes paralyses follow the convulsions. Anglade (17) reports several interesting cases of this type which came to autopsy. One of the patients had his first convulsion at sixty-three and lived to seventy-one. According to Etienne and Richard (18) the endocrine dysfunction type (non-diabetic) may be cured by proper therapy. Our own patient of this group was "cured" of her convulsions but died a year later of apoplexy. The hypertensive epileptic is always in danger of cerebral or cardiac catastrophy or terminal uremia. Anginoid attacks are not infrequent. One of our patients in this group is alive nine years after her first convulsion. Theodore C. Janeway (19) states that in his series of 7,872 cases of hypertension "onset with convulsive seizure was a great rarity." In one case, however, "four years intervened between the initial uremic convulsion and death." This was doubtless a case of hypertentive epilepsy and not of uremia. The diabetic type of senile epilepsy is now perhaps the most hopeful of all—thanks to insulin.

SUMMARY

(1) Twelve cases of epilepsia tarda are reported and a classification is suggested.

(2) A background of combined circulatory defects is demonstrable in all cases—indicating the probability that moments of deranged cerebral circulation constitute one exciting cause of the convulsions in these cases.

(3) Differential diagnosis of the types of epilepsia tarda is presented with emphasis on their separation from uremia in which generalized convulsions are rare and only terminal.

(4) Prognosis and treatment are briefly presented.

(5) A selected bibliography is appended.

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The Allergic Reaction, The Basis of Tuberculin Therapy*

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Difference in Reaction of the Non-Tuberculous and the Tuberculous To Tubercle Bacilli

Clinical tuberculosis is a disease which is usually engrafted upon an individual who has been previously infected by tubercle bacilli; and who, because of this fact, offers a special resistance to their growth and spread. If the secondary implantations are not produced by too large numbers of bacilli, the infection proves to be abortive or heals; or, if infection becomes well established, the resulting disease nearly always assumes a chronic course. That is, the tendency to proliferation on the part of the local cells exceeds the tendency to exudation and degeneration.

When bacilli first implant themselves in the tissues of a host the numbers that cause the infection are usually small. It is well that this is true; for, the only defense that is transmitted to the organism at birth is the natural defense of the tissues, enhanced by whatever degree of specific resistance may have been handed down from a race accustomed more or less to withstand tubercle bacilli. The specific allergic quality of the cell is not yet present. This is acquired later as a result of the action of bacilli and bacillary products upon the tissue cells; consequently is not available until after the host has first sustained an infection. An infection is essential to the production of a competent and lasting allergy; although dead bacilli, and possibly bacillary protein, will produce a certain degree of allergy.

Tissues of the nontuberculous possess the power of reaction to foreign bodies as is shown by the formation of tubercle. This, however, is not a specific reaction but one called forth by all foreign bodies which gain access to the tissues. The reaction of tissues to the primary inoculation of tubercle bacilli is not unlike their reaction to bits of glass, stone and other foreign material. The resemblance of tubercle bacilli to foreign bodies in this particular is largely due to their waxy content which protects them from the destructive action of the tissues.

A given infection with tubercle bacilli, overcome or rendered chronic by the host, makes him more resistant to future infections by stimulating his cells to the elaboration of a defensive mechanism. A defensive mechanism is not raised to its highest point of efficiency at one time; but is cumulative,

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increasing as metastases, from already extisting foci or from new infections from without are, successfully withstood. This defensive mechanism or immunity increases with each subsequent infection which the host is able to withstand, even though he may eventually be overcome by the disease. As clinical evidence of this immunity it is only necessary to cite the fact that while a few bacilli, becoming implanted, cause infection in one who has not been previously infected, millions and millions of bacilli may be discharged daily from ulcerations in the lung of one suffering from pulmonary tuberculosis and pass over the mucous membranes of the bronchi, throat and mouth without producing metastases. A guinea-pig receiving a second inoculation of small numbers of bacilli will live much longer than the pig inoculated only once. Experience at the Saranac laboratory shows that reinfected animals live at least twice as long as those receiving only one inoculation (1).

The chief factor which makes adult tuberculosis differ from that of childhood, and the essential factor in the successful combat of clinical tuberculosis, is this defensive mechanism developed on the part of the body cells as a result of the stimulation caused by tubercle bacilli present in infections prior to the implantation causing the clinical disease, and further stimulated by new implantations during its course.

The Nature of the Specific Defensive Mechanism Against Tuberculosis

What this specific mechanism which protects the body against tuberculosis is cannot be answered fully; yet the phenomena which have been observed in experimental animals and in the clinic give some idea of its nature. We know many things about it and have learned to recognize accompanying phenomena, even though we do not fully understand the process. While there seem to be many factors at work in protecting the organism against tuberculosis they result in two main effects: one, that of warding off reinfections or reducing their virulence; the other, that of healing existing foci.

Modern trends in immunology are following very closely the work of Metchnikoff (2) in dividing protective elements into *microphages* and *macrophages;* and the more extended studies of Aschoff (3) which have caused him to classify all cells of the body which possess the particular function of phagocytosis, likewise that of producing immunity and healing infections, as the *reticulo-endothelial system,* which he schematically outlines as given in Chart I.

The cells of the tuberculous animal possess the power to destroy the bacilli or, failing this, to surround them with an inflammatory reaction encapsulating them and preventing, or at least hindering, their spread. This is different from the non-inflammatory reaction which attends primary tubercle. This property of inflammatory reaction seems to be general in the cells throughout the body, and only awaits new implantation of bacilli to manifest itself. It is greatest, however, in tissues adjacent to tubercle and seems to vary somewhat in different tissues.

Koch (4) was the first to observe the difference in behavior of an animal

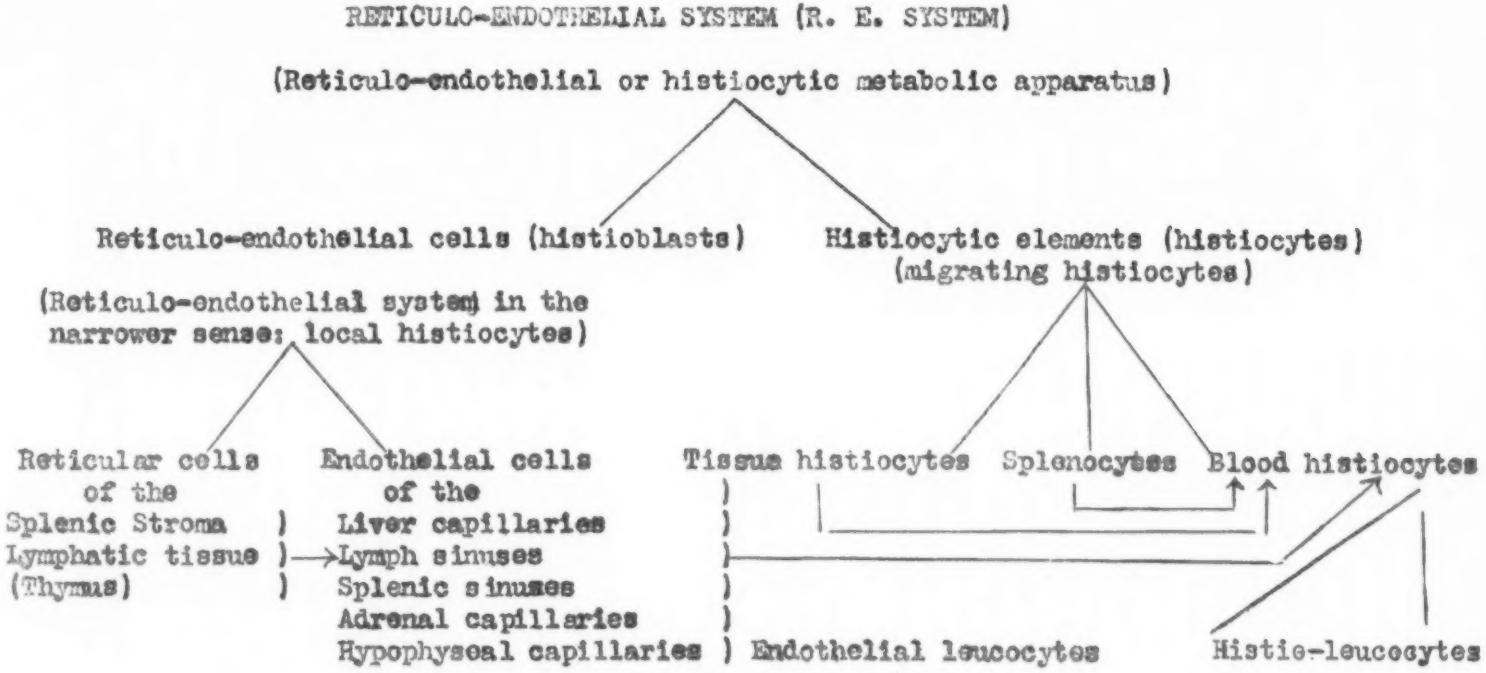
RETICULO-ENDOTHELIAL SYSTEM (R. E. SYSTEM)
(Reticulo-endothelial or histiocytic metabolic apparatus)
Reticulo-endothelial cells (histioblasts)
Histiocytic elements (histiocytes)
(migrating histiocytes)
(Reticulo-endothelial system in the narrower sense: local histiocytes)
Reticular cells of the Splenic Stroma
Lymphatic tissue
(Thymus)
Endothelial cells of the
Liver capillaries
Lymph sinuses
Splenic sinuses
Adrenal capillaries
Hypophyseal capillaries
Tissue histiocytes
Splenocytes
Blood histiocytes
Endothelial leucocytes
Histio-leucocytes

Chart I

toward a first infection and a reinfection. He noted that a first injection of bacilli into an experimental animal was followed quickly by a nodule which soon passed away, the bacilli being partly screened out in the regional lymph glands and partly passing on to the general circulation to cause infection in other organs. He observed further that after the animal was already infected a second injection behaved very differently. It produced a nodule at the site of inoculation, followed by an ulceration in a few days, which then healed, the regional lymph glands being only slightly or not at all infected. All of the elaborate studies of immunity in tuberculosis have centered about this observation.

Krause and Willis (5) and Krause (6) have studied the retarding effect of this immunizing mechanism on invading bacilli in reinfections and compared it with primary infection. They have shown that bacilli in the first inoculation of an animal will quickly pass into the general circulation while bacilli inoculated into animals which are already tuberculous pass beyond the defensive cellular barrier very slowly and only find their way into the general circulation after three or four weeks have elapsed. This is so important for the understanding of the defensive action of allergy that I will quote their findings:

"1. In normal nonimmune guinea pigs tubercle bacilli, inoculated intracutaneously or subcutaneously, are carried almost immediately (within an hour) from the portal of entry by the lymphatics.

"2. Within three or four days they have made the circuit of the body.

"3. In immune (allergic) animals their transmission is greatly retarded.

"4. They remain fixed at or near the portal of entry for about seven days.

"5. They do not reach the regional lymph nodes (superficial inguinal and axillary) until two weeks after infection.

"6. They do not become generalized to the body until three or four weeks after infection."

Many invading bacilli are engulfed by phagocytes as soon as they enter the tissues, some are destroyed and some again destroy the phagocyting cells (2) (3). There also may be a lytic action possessed by the body fluids of immunized animals which destroys bacilli. Kraus and Hofer (7) noted that bacilli injected intraperitoneally into animals suffering from tuberculosis show degenerative forms in from fifteen to thirty minutes. It would seem that there are both fixed and circulating elements in the specific defensive mechanism against tuberculosis, but that the principal reaction is cellular and not humoral.

The non-tuberculous animal injected with large numbers of bacilli is not even made ill; but the tuberculous animal suffers a severe reaction and may even succumb in from 6 to 48 hours (Koch) (4). The same effects may be produced by tuberculin. Newly born (non-infected) children have received 500 and 100 mgm. of tuberculin without causing any more disturbance in their physiologic processes than would be caused by so much water (Schreibers) (Schlossman) (8). In the infected child, on the other hand, a few milligrams would cause severe reaction.

and very large doses might even produce death.

Effect of Dead Bacilli Upon the Immunizing Mechanism

Koch (4) early observed that, when a subsequent infection took place in an animal already suffering from tuberculosis, the original foci showed signs of inflammation followed by healing. This was later confirmed by many observers. It led to the idea of attempting to cure tuberculosis by inoculations of living bacilli. To this end various types of bacilli have been tried. Many experimented with human bacilli of low virulence. Moeller experimented with certain acid-fast bacilli, particularly the timothy bacillus and the blind adder bacillus. Others used the avian and the turtle bacillus. While favorable experimental results were produced by living tubercle bacilli, the procedure was accompanied by too great danger to be used in the treatment of human beings.

Koch early experimented with bacilli which had been maintained at low temperature for a long period, also those killed by boiling and by chemicals and found that they produced effects similar to those caused by living bacilli. Sternberg confirmed this, finding that tubercle bacilli which had been killed by live steam, when injected intravenously into animals, produced the same pathologic changes that were caused by living bacilli. Caseous tubercles formed in which bacilli maintained their morphological characteristics for quite a long time.

Lowenstein (9) also confirmed the immunizing influence of dead bacilli and found a decided increase in resistance to subsequent injections of living bacilli in animals which had received previous injections of dead bacilli.

Much experimentation on animals shows that small infections produced by living bacilli, could they be controlled, would undoubtedly be of value in the treatment of tuberculosis; but there are difficulties to be overcome which cannot be estimated and dangers that cannot be controlled. Likewise, the therapeutic employment of dead bacilli, while efficacious, is impracticable because of their local necrotizing effects. Nevertheless, some artificial means of maintaining immunity at a high level and of utilizing the specific allergic reaction in producing fibrosis and healing tubercle, if obtainable, is highly desirable.

Tuberculin Stimulates Specific Defensive Mechanism

Koch early turned the search from the dangerous living and the local necrosis-producing dead bacilli to the products generated by bacilli during growth and to emulsions made from their bodies. He developed the various tuberculins which bear his name, hoping that he might find some product made from bacilli which, without carrying with it the disadvantages of living or dead bacilli, would still evoke the specific allergic reaction of the tissues, maintain immunity at a high level, and promote healing. He worked on the theory that a remedy to be efficacious would have to increase the patient's immunity and produce the same inflammatory reaction about tuberculous foci as is caused by natural infections or infections experimentally produced in the bodies of human beings

or animals who are already tuberculous. If such a remedy could be attained, by controling the dosage fibrosis could be hastened without danger of necrosis.

There are probably many factors which aid the organism in overcoming infection such as the lymph, blood and other cellular elements, particularly those belonging to the reticuloendothelial system; and the entire immunity response is favored by adequate nutrition and a well-balanced state of metabolism. It is perfectly evident that these specific allergic reactions, which are such important factors in the healing of tuberculosis, depend very much upon the maintenance of a state of physiologic equilibrium in the patient's body, as well as upon the specific bacillary stimulation itself; in fact, the immunizing response must be looked upon as an acquired physiologic reaction of the body cells. This fact is evident from the lowering or disappearance of allergy which is known to occur during periods of lowered vitality which accompany pregnancy, such infections as measles, scarlet fever and influenza, extensive active tuberculosis and other states of malnutrition and stress.

If we accept as fact that there is a specific protecting mechanism whose function is to heal tuberculosis, then we must inquire why it sometimes fails to protect the individual, and whether or not it is possible to increase its efficacy by artificial means. Frequently the infection is widespread, or it may be more limited but be very active. In either case there is great opportunity for new infections to take place caused by large numbers of bacilli, which may depress the patient's allergy and prevent a competent immunizing mechanism from being established; or the reaction may be so severe as to call out the exudative instead of the proliferative reaction. This is only another way of emphasizing the well recognized fact that tuberculosis should always be treated when the disease is limited in extent and inactive if best results are to be attained, and that advanced tuberculosis presents problems which are difficult to overcome.

We must never lose sight of the necessity of the local inflammatory tissue reaction in healing. This inflammatory reaction represents the acquired specific increased activity on the part of the body cells which is called out to aid the organism in combating the action of bacilli and their products. It limits the spread of bacilli and builds a fibrous wall about them, without which the body could not overcome infection. It is also evident that it must be more efficacious where the process is less severe and less extensive, because there is less opportunity for reinfection to occur, and such as do occur are apt to be caused by fewer bacilli and take place at longer intervals, hence cause the proliferative type of reaction, which consists of a slight hyperemia with little or no exudation.

This allergic reaction can be brought about in experimental animals by the injection of either living or dead bacilli; and when it has disappeared or is waning it can be restored by the injection of either living or dead bacilli or tuberculo-protein. Immunity can be heightened and a specific inflammatory reaction can be brought about in the tissues surrounding tubercle by the injection of preparations of bacillary protein which are of themselves unable

or barely able to produce histologic tubercle. This is the basis upon which the therapeutic effectiveness of those tuberculins rest which do not contain bacillary bodies.

It must be understood that two processes go on in tubercle simultaneously: one, caseation, in the center, destructive in character; and, the other, fibrosis at the periphery, conservative in character. Even though caseation is present in the center of tubercles, if the patient is able to put up a competent defense, those new infections which are produced by a few bacilli which repeatedly take place at the periphery are met by reaction which favors the production of fibrosis and the limiting or healing of tuberculous foci. Only reinfection by large numbers of bacilli is serious or dangerous. Every slight allergic reaction should increase the patient's immunizing mechanism and help to heal foci which are the subject of the reaction. Not necessarily every severe reaction, but oft-repeated severe allergic reaction has a tendency to produce necrosis in existing foci and favor dissemination of bacilli. The dose of tuberculin, however, unlike the natural spreading of tuberculosis in the host, is under control, and there is no reason why a reaction of dangerous proportions should ever be brought about if the physician understands the pathology of tuberculosis and the effect of tuberculin in causing the allergic reaction. Therein lies the value and the safety of the remedy.

With our recent comprehension of the allergic reaction and its part in the picture of tuberculous disease (Pottenger 10 and 11), as far as its character and course, likewise its healing, are concerned, tuberculin should gradually come into its own and be recognized as a measure for taking advantage of the healing properties of the allergic response.

While tuberculin, in which I include all products containing bacillary protein, has not assumed the place in therapy that its specific action warrants, yet it must be recognized as having an action greatly desired in the treatment of the sluggish infiltrations and ulcerations of tuberculosis which though, at first, limited in extent do not heal because the necessary stimulating reinoculations fail to appear. The efficacy of this allergy stimulating action of tuberculin may be best studied in such visible local lesions as those of the eye, larynx, lymphatic glands, testicle, tongue and skin, although they are no more convincing than the favorable changes which appear in the carefully observed lung.

Ocular Tuberculosis. Tuberculosis of the eye is one of the most satisfactory of all tuberculosis lesion to treat. v. Hippel (12) reported in 1905 on 240 cases of ocular tuberculosis of all forms treated with tuberculin and showed healing in 75.7 per cent and improvement in 17.3 per cent more. While this was a far greater percentage of favorable results than ever had been produced by other methods of therapy, yet it was not sufficient to make tuberculin the remedy of choice in ocular tuberculosis because of the prejudice which was created against the remedy as a result of its wrong employment during its early period of trial; nor was it sufficient to stimulate those who treat tuberculosis of the eye to familiarize themselves with its use so

as to be able to use it effectively. My own experience in ocular tuberculosis has been limited to about a dozen cases, and my results have been nearly all satisfactory. I recently saw a patient whom I had treated successfully for tuberculosis of the iris eleven years ago. She had no recurrence in the meantime.

Larynx and Tongue. The larynx and tongue also present visible lesions which are convincing of the healing effects produced by tuberculin. I have observed hundreds of cases of tuberculosis of the larynx during tuberculin treatment and have noted the salutary effect of focal reaction in its influence upon the healing process. I have noted the same in tuberculosis of the tongue The prognosis in tuberculosis of the tongue and larynx is favorable, unless dysphagia is present to such an extent as to interfere with nutrition, or, an accompanying pulmonary lesion is too extensive and too active.

Adenitis and Tuberculosis of the Testicle. Glandular tuberculosis and tuberculosis of the testicle are lesions that may also be watched during their treatment and are most convincing in their response.

Pulmonary Tuberculosis. My experience in the treatment of pulmonary tuberculosis now comprises several thousand patients, 75 per cent of whom were sanatorium cases and were closely observed. The treatment and after observation of these patients has extended over a period of thirty years. As a result of this experience I am convinced that the chances of cure in pulmonary tuberculosis can be improved greatly by the administration of tuberculin; that it not only increases the patient's chances of securing a healing but decreases his liability to a recurrence of the disease. This would be expected from its property of stimulating the allergic reaction to continue building up encapsulating fibrosis, after the stimulating natural reinfections have ceased to take place.

Tuberculin Reaction

As has been emphasized throughout this paper, an essential factor in the healing of tuberculosis is the focal reaction. Focal reactions produced by reinfection, inoculation of dead bacilli and tuberculo-protein are all the same. Slight reaction produces most of its effect in the periphery of the tubercle, increasing fibrosis and favoring encapsulation. Severe reaction, on the other hand, may involve the entire tuberculous focus and produce a severe exudation with necrosis. Natural reinfection in which only a few bacilli take part produces a fibroid form of tuberculosis with little exudation. This gives us the clue to the intelligent use of tuberculin. We must aim to secure the same effect as that produced by reinfection with few bacilli and avoid the type of reactions that are produced by reinfection with few bacilli and avoid the type of reactions that are produced by large numbers of bacilli (Pottenger 13).

That tuberculin is capable of doing harm as well as helping cannot be gainsaid. This is self-evident from the fact that it is capable of producing varying degrees of stimulation of the tuberculous focus, the same as accompany reinfections with varying doses of bacilli, but, if the clinician is conversant with the action of the remedy and understands the pathologic

process which he attempts to treat, danger may be avoided and only beneficial effects may be produced.

Summary

1. The natural healing of tuberculosis is brought about by stimulating a specific immunizing mechanism, so that: (1) implantations of bacilli become difficult or are prevented entirely; and, (2) the inflammatory allergic reaction is brought into play in such a manner as to surround tuberculous foci with an encapsulating wall.

2. The specific immunizing mechanism may be called out by either living or dead bacilli, and when immunity is once established it may be increased by living or dead bacilli and bacillary protein (tuberculin).

3. The allergic reaction may be taken as a measure of the specific defense in an infected organism.

4. The allergic inflammatory reaction about tubercle is necessary to healing, and can be artificially stimulated by tuberculin.

5. In the administration of tuberculin one can at will produce focal allergic reaction instead of depending on the haphazard stimulation of reinfections; and can carry them on after reinfections have ceased to take place, thus producing a more complete fibrosis and encapsulation of the bacilli; accordingly healing is hastened and the likelihood of relapse is diminished.

6. Slight reactions are necessary to healing. Occasional severe reactions may do no harm but are usually to be avoided.

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Congenital Hypertrophy, Involving the Right Side of a Female Aged Six

By E. G. Wakefield, B.S., M.D., *Brooklyn, N. Y.*

DURING the last decade the group of congenital anomalies which are characterized by an overgrowth of a single part, parts or the whole half of the body has received considerable space in medical literature. Too much has been said about the rarity, too many fanciful theories have been put forward to explain the etiology and too little has been said about a differential diagnosis for the most of us to have a clear conception of the anomaly and its accompanying conditions.

The statement has been made that this is the rarest anomaly known to medicine. This statement would be hard to prove or disprove but it is thought that the condition is not as rare as the literature would lead one to believe. The instances of the anomaly which are recorded are almost universally of extreme degree of a symmetrical development or are accompanied by a skin condition, a limp or something which has directed attention to the part. The literature shows that during the past thirty years, since Lablanc's report in 1897, the number of cases reported are two times as many as were reported during the sixty years preceding—an increase of about two hundred per cent. Of course the number of cases reported are too small to draw any conclusions however, the increase is probably due to the fact that the anomaly is generally better recognized now than formally and the incidence is not greater. The following case report supports the belief that many of these patients pass unrecognized or if the anomaly is called to our attention it may be dismissed with some such diagnosis as lymphedema.

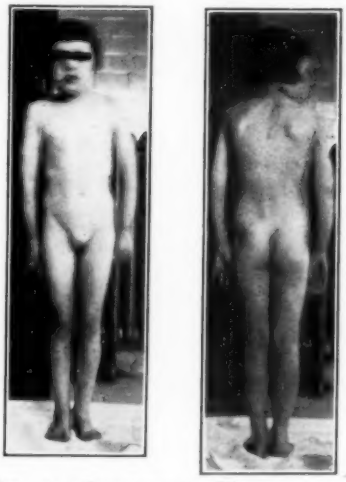

Fig. 1. Congenital Hypertrophy

Case Report

The patient, an English girl aged six. She was born by a face presentation. Labor terminated spontaneously within about twelve hours. Immediately after birth she cried and seemed perfectly normal except for the swelling of the face which is inevitable. While the face was swollen the mother noticed that the right side was the larger and the right arm was the longer. The at-

tending physician assured the mother that the child was normal and that all the swelling would subside in a fortnight. The swelling of the right side of the face persisted and before the child was a year old she had been seen in several clinics in England. Everyone who saw the child expressed an opinion that the asymmetry would be outgrown in due time. The mother was repeatedly told that the child was afflicted with lymphedema. By the time the patient had reached her fourth year she had been seen in a few clinics in the United States and the mother received the usual assurance that the child would out grow the asymmetry. When the mother inquired about the disproportion in the size and length of the arms no one seemed to be willing to commit themselves.

The patient's father and mother have enjoyed perfect health. She is the eighth child of a family of nine children who are living and well. No other congenital anomalies had occurred in the family.

In the past she had enjoyed unsually good health. Chicken-pox is the only illness the mother could recall which had incapacitated the child in the least. The growth, except the right side, and development have been normal. By the end of the first year she could walk and speak a few words. During the past year the patient has attended kindergarten and received a prize for her good behavior and progress.

Except for the right side of the body a general physical examination showed a well developed and a well nourished child of about six years. The right side of the face and the right arm were developed out of proportion to the left. The right arm appeared the longer and the right side of the thorax appeared the larger. Height 122 cm., weight 23.6 Kg., the pulses, respirations and temperature were normal. No differences in temperature, sweating, texture of skin, growth of hair and nails or in the development of the teeth could be ascertained on the two sides. She was alert, polite, co-operative and well behaved. The pupils were equal in size, regular in outline and reacted promptly and correctly. On palpation of the tissues of the right side of the face nothing unusual could be felt the only difference in the two sides was the size. No suggestion of edema could be elicited. The heart, lungs, abdomen, genitalia, peripheral and central nervous systems presented nothing unusual. Comparative measurements of the arms were as follows:

	Right	Left
Circumference of the arm at the insertion of the deltoid	17.5 cm.	15.0 cm.
Circumference of the arm 4 cm. above olecranon	18.0 cm.	15.7 cm.
Circumference of the arm 5 cm. below olecranon	16.5 cm.	15.5 cm.
Length from 5th dorsal vertebra to acromion process	14.0 cm.	12.0 cm.
Length from acromion process to elbow	28.5 cm.	26.0 cm.
Length from elbow to styloid of ulna	14.5 cm.	14.0 cm.
Total length of arm measured in tailor fashion from the 5th dorsal vertebra to wrist	56.5 cm.	52.0 cm.

Roentgen ray examination of the bones of the thorax, scapula, clavicles and bones of the arms and face showed nothing unusual. There were no demonstratable differences in the measurements of the bones from the roentgen ray plates; of course, such measurements are difficult and often inaccurate. The right leg measured a little larger than the left but the differences were so small that they were considered within the normal limits of variation. No asymmetry or deformities in the bones of the cranium could be made out. The Wasserman reaction was negative.

In making a diagnosis of congenital hypertrophy the first thing to be determined is there a hypertrophy of the large side or is there an atrophy of the small side. This may be very difficult at times. To illustrate, the late Dr. George Carpenter (2) showed a case of congenital hypertrophy to the Society for the Study of Diseases of Children in 1905 and he refused to commit himself as to whether the large side was hypertrophied or the small side was normal or atrophied when he was questioned.

Atrophy of the face, arm, leg or total hemiatrophy following anterior poliomyelitis, hemiplegia, nuclear lesions, muscular dystrophies or sympathetic paralysis can ordinarily be ruled out by the history and physical examination. If there still remains a question of nerve involvment electrical reactions of the nerves to the parts may be invaluable.

Congenital or acquired disease of the lymph drainage from the part or parts may be confusing. Presistent hereditary edema of the legs or Milroy's disease is an example of this type of affliction. In Milroy's (3) disease the edema varies from day to day and is strictly limited to the legs. Edgeworth (4) described a familial edema which is generalized, occurs in infancy and is ordinarily symmetrical. These patients die during the first few months of life. In countries where filariae abound and elephantiasis from filariae is common, this condition must be considered. Appropriate laboratory examinations will be of much aid even in chronic infestations. The history and a period of observation should be all that is necessary because filariasis is most common in adults and no asymmetry has existed since birth. Instances of the so-called "sporadic elephantiasis" which are non-parasitic in origin usually follow an injury or a surgical operation. Here the question arises, is the case reported above an example of sporadic elephantiasis? There is a history of injury of the face during birth. The points in favor of this being congenital hypertrophy not elephantiasis, lymphedema or some anomaly of the lymph drainage are; the asymmetry has maintained a normal relation during the six years of growth, there has never been any periodical fluctuation in the size of enlarged parts, the skin is normal in texture and there is no evidence of thickening, there is no edema and the arm on the afflicted side is the larger and the longer. The arm did not receive any injury during birth. Edema, which is the result of congenital or acquired disease or abnormality of lymph drainage, begins in one place and spreads from there to adjacent parts and frequently there is a daily fluctuation in the amount of swelling and practically always thickening of the skin.

The rare condition known as facial hemiatrophy (5) which is characterized by a wasting of the soft tissues and bones of the face may be impossible to differentiate from facial hemihypertrophy if the process is not advanced to the stage that atrophy of the bones of the face can be shown in roentgen ray pictures. In this condition the asymmetry may be most striking as the atrophy stops exactly in the midline. Time is the most valuable differential factor if the history is not reliable, for in the late stages of hemiatrophy there are atrophic skin changes and falling of the hair. Other rare conditions which might be confusing are: scleroderma and sclerodactyle dystrophy (6). Both of these conditions are diseases of middle life and are accompanied by atrophic skin changes (7).

Congenital wryneck may be confusing in patients who have reached adult life. All that is necessary to remember about wryneck is that the tilting of the head in a fixed position during the period of growth will enevitably produce permanent deformity.

Some writers have included in the domain of congenital hypertrophy a certain group of hypertrophies which are congenital and are associated with tumor formation. Whether these asymmetrics should be classified as true congenital hypertrophy no one is prepared to say in the present state of our knowledge. To illustrate, Froelich (8) classified congenital hypertrophies as follows: simple hypertrophy, hypertrophy resembling generalized edema, and the increase in size which is congenital and due to tumor formation. Many writers have given different classifications. It is practically impossible to make a classification which is at all valuable. This fact is clearly illustrated by Campbell's (9) case which combined the features of Froelich's first and third classes namely hyperthrophy and increase in size due to tumor formation. Instances of von Recklinghausen's disease might, if congenital, be difficult to exclude. However, instances of congenital von Recklinghausen's disease as reported by Harbitz (10) presented very little asymmetry.

Congenital hypertrophy is most commonly seen on the right side which is true of any anomaly. All sorts of skin lesions have been described as accompanying the hypertrophy. The skin disturbances are practically always vascular in origin. Mental deficiency, which was first fully considered by Greig (11) is present in from 15 to 20 per cent of the cases reported in which psychiatric examinations were made.

In a previous communication (12) some of the theories which have been advanced to explain the anomaly were enumerated. May it suffice here to say that none of the theories up to the present time will explain the partial, the total and the crossed hypertrophies. A working theory should explain all the various forms of a disease or it should exclude the forms which it does not attempt to explain. Practically all the exponents of the many theories on this anomaly, except one, (13) have been content to advance their theory and then silently repent. In the future when fashionable medical thought turns to something more obscure than embryology we are sure to have more interesting theories on this anomaly.

SUMMARY

1. An instance of congenital hypertrophy is reported.

2. A brief discussion of the diagnosis is given.

3. The anomaly is probably more common than the literature indicates.

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The Saline Wheal Test as a Measure of The Blood Supply in Arterial Disturbances of the Extremities*

By Walter G. Stern, M.D., *Cleveland. Orthopaedic Surgeon to the Mt. Sinai and St. John's Hospitals, Cleveland*

IT IS not my intention of entering into any polemic debate or theoretic discussion as to the nature of the diseases I intend to discuss or the physiology of the tests I am about to recommend as reliable guides to the sufficiency of the circulation.

The commonest symptom an orthopaedist has to deal with is pain in the toes, feet, ankles, calves and knees. After ruling out the evident cases of flat foot and other gross deformities, foot strain, bone, joint and nerve diseases, there remains, at least in our experience, a large number of cases whose only objective signs of trouble are more or less cold, clammy, cyanotic feet, blanching readily on assuming a horizontal or vertical attitude and quickly becoming cyanotic again on hanging down. This discoloration may take on many forms, it may be mottled, interspersed with patches of local anaemia, or with a sharp line (pseudo line of demarcation) about the toes just where the vamp of the shoe impinges.

In the old, such diseases commonly are arterio-sclerosis, arterio-fibrosis, arterial spasm in Bright's disease and high tension cases, atheroma due to diabetes etc., general arterial calcification (often seen only or as a beginning in the vessels of the leg), aneurism and tumor.

In younger people there are the various forms of angio-trophosis (vasomotor neurosis) Raynauds disease, arteritis obliterans, erethromelalgia, etc., running all the way from chilblains to spontaneous gangrene. The nature and cause of these conditions are beside the question to be discussed this morning. Suffice it to say that in this day of little clothing and low shoes the milder types of trouble have become very numerous.

The pulse in the dorsalis pedis artery may or may not be absent. If absent then the case is fairly easily demonstrable as a serious interference with the blood supply of the limb; if the pulse is only altered in size it would be difficult to convince an unbiased observer of much circulatory change, while if the pulse apparently unaltered although the examiner might be absolutely convinced that he is dealing with a similar case he would be lacking all objective evidences of proof.

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Up to the present time but few good and practical methods of measuring the amount of the circulation have been devised. The most accurate of these is the calorimeter method in which a limb of known size and temperature is immersed for an arbitrary length of time in an adequate amount of water of known temperature. With suitable precautions against warming this water from the outside it can readily be deducted that the relative rate and amount of the increase in the temperature of this water must come from the blood circulation in the limb and thus a test for the sufficiency of the circulation.

One of the very best of the calorimeters is that of George Neil Stewart, Professor of experimental medicine at the W. R. U. this city, who assures us that the calorimeter method is so susceptible of error from outside influences and so delicate in its manipulation that it is only to be relied on when the test is performed in the physiological laboratory and is not in any way a test suitable for hospital or office use.

The hypodermic pyrometer method of Dr. Barney Brooks of St. Louis, depends upon the ingenious method of carring the proper wires into a medium sized hypodermic syringe needle. After plunging this into the muscles of the limb to be tested and the normal temperature measured upon the galvonometer, a tight tourniquet is applied and the blood flow stopped. "The limb rapidly looses heat and when the bottom of the curve had been reached, the tourniquet is released and in a normal case this is followed by an immediate and rapid rise of temperature to its former level. In the tissues of an extremity in which there is impairment of the arterial blood supply the release of the tourniquet is not followed by an immediate and rapid rise of temperature. In any disease in which there is obstruction of the large arteries there is an appreciable period of from one to ten minutes elapsing between the removal of the tourniquet and the beginning of the rise in temperature of the tissues to be tested. In conditions of marked arterial obstruction the application and removal of a tourniquet may cause no temperature changes in the distal tissues. By the application of this method to the tissues of various levels and the distributions of main arteries it is possible to obtain valuable knowledge as to the condition of the arterial circulation of the extremity." We have not used this method because of the inherent inaccuracies of such small and delicate pyrometers and also because we do not feel justified in either making a small wound (not a harmless procedure in some of these cases by any means) or to apply a tourniquet (for so long a time.)

The direct injection of radiopaque substances into the arteries and to observe their progress by means of the fluoroscope does not appeal to my surgical judgment to be permissible in human beings suspected of having arterial diseases or obstructions, but it may well be that I am too conservative.

The oscillometer of Pachon seems to be a fairly reliable indicator of the pulsation of the artery at the level examined. Its weak points are that the instrument is forever out of order—and usually at the most critical times, cannot be used over or near bony points as

in foot, at knee or ankle, and depends for accuracy upon the patient's ability to lie perfectly quiet, which some old people with arterio-sclerotic gangrene seem to be unable to do. We have relegated it to be a check up upon the salt injection method and found but few discrepancies between them.

Following out the experiments of McClure and Aldrich who used intracutaneous salt solution injections for the purpose of studying edemas, Dr. M. B. Cohen of the medical staff of this hospital noted that while the appearance, size and shape of the wheal was more or less constant, the disappearance time varied in different individuals, and at times in different levels of the limb under observation. He conceived the idea that the disappearance time depended on the sufficiency of the arterial supply, and, since our orthopaedic department, as before said, was vitally interested in this condition and was checking up all the cases it could gather, we co-operated and examined jointly or independently over two hundred cases, and have drawn the conclusion that this method is entirely reliable and is the most convenient and simplest guide to the sufficiency of the arterial circulation of a limb. The physiology of this test is not for a mere orthopaedic surgeon to discuss.

The test is performed as follows: By means of a tuberculin syringe and a very fine needle, 0.2 cc. of 0.85 per cent salt solution is injected intracutaneously. The eye of the needle should be visible through the outer layer of the skin when the injection is made. The first injection is made at the base of the great toe, and similar ones are made at 4 inch intervals up to the leg and thigh. The sense of touch is used to determine the disappearance time, as the vasomotor changes produced by the injection often render visual judgment unsatisfactory. Normally, sixty minutes or more is required for the complete disappearance of the wheal produced by the injected fluid, though readings as low as thirty minutes at the base of the great toe have been considered normal (as we have one such reading in a patient without clinical evidence of vascular disease); but as a general rule sixty minutes or more may be considered as the normal disappearance time. In cases in which the circulation is impaired the wheal disappears in less than this, in threatened gangrene, often as quick as one to five minutes.

We could cite many cases to support our contention and show the clinical value of this test. Only a few will be in order.

Case I.—A man, aged 38, stated that he had frozen his left foot while driving an open automobile in zero weather and was taken to a hospital, where the great toe was amputated at the metatarsophalangeal joint, about three months before he first came under our observation. There was an unhealthy looking ulcer over the head of the metatarsal bone. The pulse in the dorsalis pedis artery was missing. The foot was cyanotic and cold. A diagnosis of local arterial thrombosis due to frost-bite was made, and under appropriate treatment the ulcer was soon healed.

Two years later (December, 1925), he again "froze his foot" under similar conditions, and his little toe was amputated as an emergency measure. This stump also did not heal.

Three months later, the outer border of the foot, representing the area of skin sur-

rounding the metatarsal of the little toe, became black and gangrenous, and intense pain and suffering followed. Under the same diagnosis—local arterial thrombosis from frost bite—an amputation just below the calf of the leg was proposed and was to have been carried out. We were unexpectedly called for the second time at this point. No oscillometer was handy. With saline solution and a hypodermic syringe, we performed the intracutaneous saline test on both limbs.

The right leg showed a moderate circulatory difficiency, but the left (gangrenous) leg showed a disappearance time of the solution, as shown in table 1.

TABLE 1.—Disappearance time (Minutes) of Wheals on Left (Gangrenous) Leg.

1. Just above area of gangrene..	5
2. Ankle	15
3. Lower Calf	18
4. Calf	16
5. Upper Calf.....	20
6. Knee	30
7. Thigh	30
8. Upper thigh	60 plus

A diagnosis of generalized angiotrophosis with thrombosis of the left popliteal artery was made and a thigh amputation advised. A mid-thigh amputation was performed. The flaps and muscles hardly bled at all. An organized clot was found in the popliteal artery, and one or two of the calf muscles were found already gangrenous. There was only local arterial thrombosis in the foot; from the ankle to the popliteal space the arteries were patent. Later examination showed a blood viscosity of 6.10 (Hess).

The real explanation of this case is that the man (an inveterate smoker) was suffering from angiotrophosis (thrombo-angiitis form) and that the untoward exposure to cold brought on a rapid deterioration of the circulation.

Note: In December 1925 I was again called to see this patient. He now had a beginning gangrene of the right foot. Salt test showed almost no circulation—amputation had to be performed two weeks later to save the patient's life.

Case II.—A man, aged 51, stated that two years ago he lifted a weight of about 100 pounds, helping with the knee of the right leg, and while pushing upward felt something give way in the left foot, on which he was standing. He was soon unable to put any weight on the foot, which later became discolored and swollen and was treated by a competent physician for an acute strain of the plantar fascia. From that day on he could not work on account of severe pains in the left foot and ankle; the condition was much worse in winter; the feet at times turned blue. Roentgen-ray examination of both feet was absolutely negative; the man was a distinct hypopituitary type, with bilateral knock knees and flat foot. He weighed 206 pounds (93.4 Kg.) and was 5 feet 8 inches (173 cm.) tall.

Physical examination was entirely negative for signs and symptoms produced by injury. The feet were cold, rather livid, and blanched easily when the legs were elevated to an angle of 45 degrees, and the pulses of both dorsalis pedis arteries were barely palpable. The systolic blood pressure was 100; diastolic, 89, and the blood viscosity, 6.7 (Hess). The oscillometer showed a marked diminution in the swing over both ankles and calves; it also showed that the circulation in the right leg was just as insufficient as that in the left. The intracutaneous saline test showed the disappearance time diminished in both legs, as given in table 2.

In this case, through the combination of the viscosity and the intracutaneous tests, we were positively able to diagnose a case of thrombo-angiitis, which had been masquerading for several years under the diagnosis of a purely traumatic condition.

TABLE II.—Absorption Time (Minutes) in Case 2.

	Right	Left
1. Base of great toe....	14	13
2. Middorsum of foot..	16	15
3. Ankle	16	14
4. Lower Leg	25	32
5. Calf	36	37
6. Upper Leg	60 plus	60 plus
7. Thigh	60 plus	60 plus

Showing value of a negative test.

Case III.—A man, aged 45, worked steadily until three months ago, when he dropped a heavy weight on the great right toe and fractured the first phalanx. This was treated by rest and splints. Since the accident, this foot had been cold and weak, and he could hardly walk on account of pain and weakness in the limbs. The intracutaneous saline tests taken in the course of the differential diagnosis showed a disappearance time of over sixty minutes in all parts of the thighs, leg, calf, ankle and foot, and a circulatory disturbance was ruled out. The cause for his condition proved to be a beginning lateral sclerosis.

Case IV.—In a man, aged 27, with bilateral thrombo-angiitis, the right foot was apparently much worse than the left. One toe of the right foot was ashy gray, but not gangrenous. The patient had had typical attacks of intermittent claudication, and the foot blanched on an elevation of 45 degrees. The intracutaneous saline test showed the circulation of the left side to be more deficient than the right, as will be seen from table 4. In order to check up the correctness of this test, the oscillometer was used by an intern who had no knowledge of these findings; he found a marked loss in the swing of the needle on the left side and a better reading on the right, thus checking up this unexpected finding to a nicety.

TABLE III.—Absorption Time (Minutes) in Case 4.

	Right	Left
1.	22	10
2.	45	25
3.	48	29
4.	24	31
5.	36	35
6.	39	36
7.	36	38
8.	41	37
9.	48	42

SUMMARY

1. In the absence of edema, the intracutaneous salt solution test is a simple, rapid and accurate method of determining circulatory deficiencies in the extremities.

2. Sixty minutes or more is the normal disappearance time of the salt solution.

3. In all instances in which clinical circulatory deficiency exists, the disappearance time is diminished; in the area just above the seat of a gangrene (existing or threatened), it is frequently as low as five minutes.

The Mitral Heart Roentgenologically*

By David Steel, M.D., *Cleveland, Ohio*

IN ORDER to understand the changes in the cardiac silhouette under pathological conditions, it is, of course, quite necessary to know the normal. If you will bear with me for just a moment we will go over briefly the normal outline in the various positions.

On the right side in the A. P. position we notice usually two arches—the lower, larger arch being that of the right ventricle and the upper, less pronounced arch—that of the ascending portion of the aorta. In addition to these there can occasionally be distinguished two more. With a low diaphragm, and especially in the emphysematous, there is sometimes seen a small outward, concave shadow running downward in the cardio-diaphragmatic angle. This is the shadow of the inferior vena cava. The other shadow runs upward from the aortic shadow parallel to the spine save in its upper portion where it arches outward in the region of the clavicle. The parallel portion is regularly referred to the superior vena cava. It is less dense than the aortic shadow and can be differentiated fluoroscopically by a difference of pulsation. The upper arching portion of this shadow is cast by the right innominate vein.

The left side in the A. P. position shows four arches which from above down are (1) aortic knob, (2) pulmonary artery, (3) appendage of the left auricle and (4) the left ventricle. In many cases the middle two are fused to form one—the so-called mid-cardiac shadow. Notice that below this the prominent left ventricular shadow forms an incisura—occasionally, and by far more common in children, we see a fifth left arch which occupies the left cardio-diaphragmatic angle. This arch is concaved outward and is ascribed by Groedel to adhesions of the pericardium to the diaphragm.

The right ventricle does not form a part of the silhouette in the A. P. position. It lies entirely anterior. A portion can sometimes be seen through a large gas bubble in the stomach.

Considerable knowledge is added by the oblique positions. In the first oblique position—i. e. the ray passing from the left posterior shoulder to the right anterior—quite a change takes place.

We now have an anterior and posterior border with a normal clear space behind the retro-cardiac or Holzknecht's space. Anteriorly (1) we see the aorta, (2) pulmonary artery (3) a small portion of the right ventricle (the amount depending on the rotation), (4) the left ventricle. Posteriorly we have from the above downward (1) a cross section of the pulmonary veins, (2) the left auricle, (3)

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the right auricle and (4) the inferior vena cava. Notice particularly the position of the left auricle and pulmonary artery.

In the second oblique view i.e. the ray passing from the right posterior to the left anterior—we see anteriorly (1) the ascending aorta—the best position for the study of the root. (2) a narrow portion of the right auricle and (3) the right ventricle. The greater the rotation the more is this arch formed by the right ventricle and the less the rotation the greater is it formed by the right auricle.

Posteriorly we see (1) the left auricle and the left ventricle. Notice in this view the positions of the right ventricle and the left auricle. In this position the rays run perpendicular to the aortic arch and we, therefore, have the classical position for the study of the arch and root. The descending aorta runs down along the spine shadow. This is also the classical position for the study of the left auricle, which sometimes can best be seen in the inverse second oblique.

The left auricle in mitral disease is the first to undergo dilatation. Due to congestion in the lesser circulation, and sometimes also to a sclerosis of the pulmonary artery, the right ventricle hypertrophies. It is the changes in this chamber that are responsible for a large proportion of our changes in the silhouette in mitral lesions. The right ventricle is situated anteriorly against the sternum and inferiorly against the diaphragm.

When enlargement takes place it can not go forward or downward and the result is a cardiac rotation. By this rotation the heart becomes more erect, the left ventricle goes more posteriorly, the pulmonary artery comes more anteriorly and to the left, the left auricle is rotated further posteriorly and the aortic knob goes posteriorly and becomes partially hidden by the spine shadow. Stasis accounts for an enlarged pulmonary artery and left auricular appendage. These two structures will then fill out the cardiac incisura. The right auricle is not only made more prominent by pressure from the enlarged right ventricle but is enlarged because of stasis.

We have then in the A. P. position the following changes:—

(1) Erect or oblique heart.

(2) Prominent left auricular appendage.

(3) Prominent pulmonary artery.

(4) Insignificant or absent aortic knob. (Not only does rotation decrease the prominence of the aorta, but it is actually smaller due to habitual scanty filling.)

(5) High junction of the pulmonary artery and aortic knob.

(6) Elongated prominent right lower arch of the right auricle.

Additional information is added by a study in the oblique positions. Remembering that normally the left auricle is for the most part posterior, that it is rotated further posteriorly in mitral disease and that we have an enlargement, it is easy to explain the encroachment of the mid-portion of the retro-cardiac space in the first oblique view.

Normally this space is clear, but in mitral disease the auricle obliterates the mid-portion, leaving a clear space above and characteristically a clear, triangular area below. The enlargement in mark-

ed cases will displace the oesophagus to the right. In the second oblique view we not only make out a definitely enlarged left auricle but we can form some idea of the right heart as indicated by the increase of the arch. We can therefor add to the above:—

(7) Obliteration of the mid-portion of the retrocardiac space with a characteristic light triangle in the lower portion.

(8) Displacement of the oesophagus by the dilated and hypertrophied left auricle.

(9) An increase in the prominence of the right lower arch in the second oblique view.

In doubtful cases the hilus region is a definite aid.

We must grant first that the hilus region is formed mainly by the vessel shadows, the other structures forming only a minor part. Both hilus regions are increased in mitral lesions. The left side is obscured by the heart shadow, but the right side can usually be distinctly made out. In about one third of the cases the main branch of the pulmonary artery stands out sharply, being contrasted on its external side by the bright lung fields and on its internal side by the descending main branch of the right bronchus.

If measured at the level where the artery crosses the bronchus it will be found normally to vary between 11-14 mm. the average being 13. A measurement above 14 mm. therefor indicates some increased pressure in the lesser circulation, a fact which has some weight in questionable cases. We then have

(10) A change in the hilus region.

The above description is that of a so-called mitral configuration. Often times when clinical findings suggest either a stenosis or insufficiency we find roentgenological evidence of both. The silhouette of pure stenosis shows no increase in the region of the left ventricle and the region of the left auricle is usually more marked. With an insufficiency we have a definite enlargement of the left ventricle as indicated by an increase of the left median distance. With enlargement of this chamber we have all four chambers involved and the result is a ball shaped heart. Also with an insufficiency the cardiac incisura is partially restored by the enlarged ventricle and the left auricular appendage does not appear as marked.

In summary we then have as signs of mitral disease:—

(1) Erect or oblique position of the heart.

(2) Prominent left auricular appendage.

(3) Prominent pulmonary artery.

(4) High junction of the pulmonary artery and aortic knob.

(5) Insignificant or absent aortic knob.

(6) Elongated lower right arch joining the ascending aorta higher up than normally.

(7) Obliteration of the mid-portion of the retro-cardiac space leaving a characteristic light triangle in the lower portion.

(8) Displacement of the oesophagus of the enlarged left auricle.

(9) An increase in the right lower arch in the 2nd oblique position.

(10) Changes in the hilus region.

(11) A ball-shaped heart in cases of mitral regurgitation.

Studies in Rickets*

By H. J. Gerstenberger, M.D., J. I. Hartman, M.D., John D. Nourse, M.D., Norman C. Wetzel, M.D., D. N. Smith, *B.S.*,
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INSTEAD of attempting to go into detail concerning the etiology, pathogenesis, pathology, symptomatology, prognosis, treatment and prophylaxis of rickets, I prefer to use this opportunity to acquaint you with some of the work which has been done at the Babies and Childrens Hospital by Drs. Nourse, Hartman, Wetzel and myself, in connection with human rickets.

Let me indicate too, that while progress has been made in the accumulation of knowledge concerning the etiology and treatment of rickets, but little has been accomplished in fathoming the pathogenesis of this disease. However, some progress also has been made in this direction. It is an accepted fact, for instance, that the inorganic phosphate content of the blood serum of rachitic infants is distinctly lowered, and that the glycolytic power of the blood is likewise reduced. In some phases of rickets the calcium level is but slightly below normal; whereas in others it is decidedly so. Whether the reduction of the glycolytic power of the rachitic blood is a result of the lowered inorganic phosphate content of the serum or whether both characteristics are due to the same cause is not known. Nor is it even agreed that the changes found in the bones are secondary to the changes in the blood serum—some authors maintain that the characteristic changes in the blood serum develop only when rickets has reached a severe grade; some that all bone changes are dependent upon condition of the blood serum, and some that both abnormalities appear more or less at the same time, and are simply synchronous manifestations of the same disturbance.

Today, rickets is considered to be a metabolic disease affecting the body as a whole. Its clinical picture, however, is the result of pathological changes produced by it principally in the osseous system, the muscular system and the nervous system.

Its prime and basic cause is the inadequate exposure of the human body to the actinic rays. The type of diet, in our opinion, plays a secondary role; the proof of this latter statement are the following facts:

1. Exposure of the human body to the ultra-violet rays will cure rickets no matter what the diet may be.

2. Undiluted cows milk has been known by pediatrists to be rather an aid to the development of rickets than to its prevention.

3. Human milk, the ideal food for

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the human infant, can not be depended upon to protect the human offspring against rickets and will not cure this disease even though the mother ingest potent cod liver oil in the dose of one tablespoonful daily.

4. Human milk from a mother whose skin has been adequately exposed to the ultra-violet rays, cures active non-healing rickets in human infants.

In other words, the natural and primary combination for the human infant is actinic ray and skin, and not diet and gastro-intestinal tract.

During 1925 and 1926 we gave to three cases of active non-healing rickets, breast milk obtained from mothers or wet nurses to whom were given daily one tablespoonful of cod liver oil, beginning at least three weeks before the milk was ingested by the infants. In two cases the milk was canned, and in one case the milk was fresh, that is, it was taken directly from the breast. In all three cases no effect could be demonstrated either in the blood or in the X-ray.

We chose cod liver oil because we felt that it was a simpler method of administering the anti-rachitic factor to the mother than would be the exposure of her skin to the ultra-violet rays. However, in view of the fact that the cod liver oil ingestion was found to be of no value from the standpoint of curing rickets in the human infant, a finding which corresponded with results obtained by Alfred Hess in rats, we decided, especially in view of the fact that it was reported by Steenbock for goats and by Alfred Hess for human mothers that exposure of the integument to the actinic rays enabled the mother to produce a milk with definite anti-rachitic powers, to expose human mothers to these rays and to feed their milk to active non-healing rachitic human infants. Hess fed the human milk to rats. The diets of the mothers receiving cod liver oil were just as complete as were those of mothers being exposed to the ultra-violet rays, and contained all of the essential food substances in sufficient quantities and calories.

In the two cases in which this method was used positive results were obtained. In other words, we were able to impart to human milk by exposing the skin of the mother to actinic rays, anti-rachitic properties of a degree sufficient to cure active human rickets. Just as in the cod liver oil series, one of the infants received this milk canned and the other in a fresh state.

These findings suggest the possibility that cod liver oil cures rickets in a different manner than does the actinic ray, and it again indicates that fundamentally human rickets is a disease due principally to an insufficient exposure to the ultra-violet rays rather than to the intake of an inadequate diet.

It has long been known, as was first emphasized by Wieland, that the most marked clinical signs of rickets are found in that part of the osseous system which happens to be growing most at the time when rickets is active, and McCallum, Howland and Kramer, and Cavins have shown that starvation of rachitic rats again starts calification.

Recently, we published the results of observations made by us on the prevention of rickets in premature infants. It is well known that these human beings are especially prone to the de-

velopment of this disease. The sum and substance of or conclusions was:

1. That rickets can be prevented and cured in premature infants.

2. That the amount of cod liver oil required is small; the average daily dose for the group being 1.76 cc given in the form of S. M. A. or Protein S. M. A.

The diagnosis of rickets in our work was and is based principally upon the results of the calcium and inorganic phosphate determinations of the blood serum, and also on the roentgenological pictures of the wrist. Clinical examination in early cases of rickets in our experience is less dependable. No definite clinical signs were found in any of our cases.

In the study of the material which formed the basis of this report, it seemed to us that the prevention of rickets was easier when the gain in weight was relatively slow, and more difficult when it was excessively great and rapid.

In the series just referred to, two of the seven infants receiving Protein S. M. A. developed rickets. Both of these infants, however, lost their rickets even though they were continued on the same mixture and were not given more of the anti-rachitic factor in any form. These two infants gained remarkably in weight, but as they grew older, their relative gain in weight became less and the actual quantity of cod liver oil ingested, slightly greater, because of a slight increase in the food consumed. The increase in the cod liver oil intake however, was very slight, being not more than the 0.1 to 0.2 cc of cod liver oil per twenty-four hours. Consequently, it was our impression that the principal factor for the healing of the rickets in these two infants, was the relatively reduced rate of growth, and that the opposite condition, namely, the earlier excessive high rate of growth, brought about the development of rickets. One of these infants weighed 1050 grams at birth, and was one of twins. The other twin weighed 1150 grams at birth, and gained at a slightly less rapid rate, and avoided the development of a definite rickets. In all probability in this second twin rickets was on the road to be developed in a recognizable form, inasmuch as there was a drop in the calcium and inorganic phosphate figures to levels which are considered to be just within the boundaries of normal. After this very temporary drop, the amounts of calcium and of inorganic phosphate in the blood serum rose to levels that are unquestionably normal.

In searching for a basis of study to determine the role of the rate of growth in increasing the requirement of cod liver oil in the prevention of rickets in essential normal premature infants we decided to use the daily gram gain in weight per kilogram body weight.

Our impressions were corroborated by one of our group (Dr. Wetzel) who found that the infants of our series who temporarily developed rickets, received for varying periods less cod liver oil per gram gain in weight per kilogram body weight than did those who did not. For instance, the one twin who developed rickets, gained during the preceding period, from 19 to 13 grams per day per kilogram body weight, whereas the brother who did not develop definite rickets, gained

from 11:5 to 9.0 grams. Inasmuch as the intake of cod liver oil for these twins was virtually the same during the period previous to the development of rickets, the only varying factor of any importance upon which we could put our finger, was the much greater gain in weight per kilogram body weight in the sister over the brother. Evidently, age and heredity could play no important part in these twins.

On the basis of these findings, a curve was constructed, indicating the amount of cod liver oil per daily gram gain in weight per kilogram body weight, presumably necessary to prevent rickets. The relative positions of the various infants of our first series to this curve were registered, and it was evident that the infant developing definite rickets was below this curve for a much longer period than was her twin brother who had a very temporary drop in his calcium and inorganic phosphate serum figures without developing dependable signs of rickets.

We have since then used this curve as the determinant of the amount of cod liver oil to be used in the prevention of rickets, in a series of seven premature and one full-term infants. These infants were observed for periods of four to five months, which is ample time for the development of rickets in growing premature infants not exposed to the action of the antirachitic factor. X-rays of the left wrist were taken at weekly intervals; estimations of the blood serum calcium and inorganic phosphate were made 1-2 weeks after admission in four cases; 3 weeks in three cases, and 7 weeks in one case, and thereafter in every instance at periods of two to four weeks. The infants were examined at frequent intervals for clinical signs of rickets.

The maximum daily average amount of cod liver oil was 0.71 cc, and the minimal 0.4 cc; for very short periods the maximal amount of 0.2 cc and the minimal amount of 0.1 cc were given in some cases. The total amounts of cod liver oil received during the periods of observation from 117 to 154 days varied for the individual infant between 55 and 95 cc. An infant receiving the customary dose, namely 1 teaspoonful three times a day, during this same period, would have received a total of 1800 to 2200 cc, or in other words, twenty to thirty times more than we have found effective. Only one infant developed definite evidence of a mild rickets (X-ray and Blood) which properly healed on the same diet without any increase in the actual amount of cod liver oil received. The rate of growth, however, was less during this healing period and consequently the relative amount of cod liver oil per gram gain in weight per kilogram body weight was consequently greater. This infant gained in weight at a greater speed than did any other, except one, whose cod liver oil intake, however, was 30% greater. We believe this to indicate that the amount of cod liver oil used by us in this series as determined by the curve employed, is very close to the minimum amount required to prevent rickets. The possibility of such an occurrence was anticipated by Dr. Wetzel who constructed a second curve, which allows a 10% increase as a matter of safety. We at first thought that we should use the second curve as a surer method of preventing rickets. How-

ever, we chose the first curve in the anticipation of obtaining an experience such as we have just reported, which we feel is more valuable than an absolutely negative finding in every case.

From experience obtained in this second series, we have concluded as follows:

1. That rickets can be prevented in rapidly growing premature infants by the early and continued use of cod liver oil.

2. The amounts of cod liver oil required per day to prevent rickets in well infants, is small and varies directly with the rate of per gram gain in weight per gram per kilogram body weight.

Consequently, the actual amount of cod liver oil required per twenty-four hours is greater, the younger the infant. This seems paradoxical, but we are convinced that it is correct.

The cod liver oil requirement as determined by the curve used by us, gives quantities that are very close to the needed amount of cod liver oil used by us to prevent rickets. As the cod liver oil administered by us was in some form of the S. M. A., we do not suggest that straight cod liver oil in such small amounts as 0.4 to 0.7 cc per day be used in practice. Nevertheless, we believe that straight cod liver oil, if given continuously, after the first week of life, will prevent rickets in normal infants, even though the amounts be much smaller than the customary dosage. The giving of cod liver oil within one week after birth should become a routine, throughout the entire year, inasmuch as newly born infants are usually protected against the sun. We are at present attempting by the same method of study as outlined above to determine the minimal amounts of straight cod liver oil required to prevent rickets.

From two illustrative cases it will be seen:

1. That the amount of cod liver oil became less as the infant grew older.

2. That the one infant remained free from rickets throughout the period of observation.

3. That the one developing rickets lost it without any change in diet.

Mixed Tumor of Parotid Type, Orbital in Location

By Earl D. Cumming, M.D., *Cleveland, Ohio*

ON OCTOBER 17, 1921, Mrs. Minnie Beese, age 52, presented herself at St. Luke's Out Patient Department for physical examination, with two complaints:

(1.) Pain in lower right abdomen, and

(2.) A prominence, very pronounced, of the right eye.

Present Illness: For the past seven years patient has had intermittent attacks of pain in the right lower abdomen associated with tenderness on pressure at approximately McBurney's point. She has never had nausea or vomiting with these attacks, and it is this disturbance that lead her to consult a physician rather than the prominence of her right eye, which she accepted as a matter of course. About three years previously she had had profuse lacrimation from the right eye and consulted an oculist, from which time she had noticed that that eye was becoming more and more prominent. When she closed the left eye and looked only with her right eye, she became nauseated. She noticed that her vision in the right eye was becoming less and less acute.

Past History: Patient was operated upon 19 years ago for ectopic pregnancy. She had had an osteomyelitis of the right forearm and left femur when eight years of age. She had had nocturia for the past seven weeks. No abnormal amounts of urine. Menopause two years previously. She had three children; two living, one died of diphtheria, one miscarriage, and one ectopic pregnancy.

Family History: Negative.

Physical Examination: Patient was an obese woman weighing 198 lbs., and measuring 5 feet, 2 inches, in height. There was a definite right-sided unilateral exopthalmos, but the palpebral fissure on the right side was narrower than that on the left—intra-ocular pressure was normal on both sides. With the eyes rotated caudad there could be palpated under the supraorbital ridge an irregular mass, 2½ cms. long and about 1¼ cms. wide—slightly movable and painless. She was referred to the eye department, where her vision was 61/10-3 in both eyes. Her retinae were essentially negative. Both pupils were equal and active. The patient's blood pressure was 182/104. Her left cardiac border was 2 cm. to the left of the mid clavicular line. The upper cardiac border at the third rib, and the right border of the heart corresponded with the right border of the

*Presented to the American College of Physicians, St. Luke's Hospital, Cleveland, Ohio, February 22, 1927.

sternum. The aorta was slightly widened. Palpation corroborated the evidence gained by percussion as to the left border. Auscultation revealed both sounds of normal quality and intensity over the various valvular areas. There were no murmurs.

An examination of the lungs revealed a few coarse rales at both bases posteriorly. She was afebrile. Her pulse was 80. The abdomen was very thick walled and pendulous. It was very difficult to palpate the spleen and liver, neither of which were enlarged to percussion. There was some tenderness on deep pressure at McBurney's point. Vaginal examination revealed no abnormalities. Neuro-muscular examination showed all tendon reflexes, with exception of the achilles on both sides, present and active. The plantar response was normal bilaterally.

The urine showed an occasional hyaline and coarsely granular cast, and a few W. B. C's.

R. B. C.	W. B. C.		Hb.
5,840,000	5,840	90%	Tallquist

Ewald Meal: Combined acidity.....27%
Free acidity.....35%
Total acidity.....62%
No occult blood....Very few

W. B. C., numerous squamous epithelial cells present.

The spinal fluid was under normal pressure, showed 6 cells, negative Globulin and Wassermann. The blood Wassermann was negative.

The pre-operative diagnoses were:

1. Orbital Tumor, right side.
2. Obesity.
3. Chronic Myocarditis.
4. Chronic Nephritis.
5. Hypertension.
6. Chronic Appendicitis.

On the 8th of November, 1921, Dr. Simonds and Dr. Shackleton removed the orbital tumor, which was fatty in appearance, lobulated, and measured 2½ cms. by 2 cms. by 1 cm. The microscopic section by Dr. King, corroborated by Dr. Mallory was Mixed Tumor of Parotid Gland Type. Following the operation Dr. May inserted 50 mg. of radium into the incision, screened by ½ mm. of silver, 2 mm. lead, 3 mm. of rubber and this was permitted to remain for nine hours. She made an uneventful recovery except for a ptosis of her right under lid, and a retraction of her right eye ball. Up to the present moment the patient has had no recurrence of her tumor and her vision is OD 20/60. OS 20/20. Her blood pressure, urinary findings, and cardiac findings have remained essentially the same, but her weight has dropped to 171 lbs., and she feels perfectly well.

Conclusions: My only excuse for reporting this case is the unusual location of this type of tumor. In the literature I have been able to find only four cases of mixed tumor of the parotid type found in the orbit.

In the December 1926 number of the American Journal of Medical Sciences, Joseph McFarland of Philadelphia reports ninety tumors of this type which came under his observation, and collected from the literature 269 other cases, and gives a comprehensive and complete resume of the histological and embryological study of the subject. In none of McFarland's cases was the tumor found in the orbit, but usually they occurred in or around the parotid, or other salivary glands. The theory of accidental sequestration of embryonal cells during the early and compli-

cated development of the face and neck affords the most satisfactory explanation of the origin of these mixed tumors. By that theory it is easy to account for the variety of tissues found in the tumors and for their varying proportions and conditions. They are individual entities, and have no relation to the normal structures in which they occur, and from which they do not arise. He thinks that they should be called "Mixed Tumors" and nothing else. They are inherently benign, but commonly recur after excision, and if frequently disturbed become destructive and invasive without giving metastasis. The histology is extremely complex—but on that account the microscopic diagnosis is usually easy. Histologically, prognosis is very difficult to determine, and is often misleading. Malignant change whether "sarcomatous" or "carcinomatous" in mixed tumors must be rare, and its occurrence is difficult to prove. Intervals of ten, twenty, or even thirty years may elapse between operative removal of a mixed tumor and its recurrence, therefore, caution should be exercised in declaring any case to be cured.

Chronic Ulcerative Colitis

By Horace W. Soper, *St. Louis, Missouri*

UNDER the term colitis we consider the ordinary catarrhal form as well as the chronic ulcerative type. Chronic ulcerative colitis must be differentiated from tuberculosis, syphilis, post dysenteric ulcers and polyposa intestinalis adenomatosa.

Chronic ulcerative colitis has been a medical problem for many years. It has been designated idiopathic, nonspecific, post-bacillary, infectious and septic ulcerative colitis. It appears to have been described as a disease entity by Wilke and Moxon (1) in 1875 and White (2) in 1888. There has been a great deal of difference of opinion as to the etiology of the disease. Logan and his co-workers considered some metabolic disturbance as the etiologic factor. Hurst (3), Einhorn (4), Leusden (5), Thorlakson (6) and most of the German writers considered the dysentery bacilli as being the original invaders. Prof. L. Kuttner (7) and Rosenheim (8) have both reviewed the subject recently and suggested the name of chronic suppurative colitis. They believe it follows bacillary dysentery. Streptococci are thought to be the etiologic factor by Jex-Blake (9), White, Yeomans (10), Wallis (11), Hewes (12), Lockhart, Mummery (13), Rolleston (14), and T. R. Brown (15).

Logan (16) from the Mayo Clinic in 1919 gave the most comprehensive description of the disease and presented x-ray plates and pathological specimens. He concluded "that chronic ulcerative colitis is a disease of long duration. The final stage ending quickly from toxemia or perforation. Nature's efforts to cure causes excessive fibrosis with resultant deformity of the colon. The forms of treatment are thus far unsatisfactory. The best results come from dieting and giving surgical rest to the colon."

In December 1925 Bargen and Logan (17) published their article on, "The Etiology of Chronic Ulcerative Colitis." They found a diplococcus which they believe to be the primary etiologic factor. They made cultures from the lesions of sixty-eight patients suffering from the disease and found the organism in 80% of the cases. Twenty healthy colons were swabbed in the same manner and only once were diploccocci of similar characteristics isolated. Their chain of evidence appears to be complete. They injected rabbits with the brain broth culture and produced a diarrhea and lesions of ulcerative colitis. Rabbits were injected with various strains of streptococci and the lesions in the colon were not produced. In persons suffering from ulcerative colitis they isolated the diplococci in periapical abscesses and in the tonsils. The culture was injected in rabbits and dogs, producing ulcerative colitis. The organism is a Gram-positive, lancet shaped diplococcus growing in twos and fours and has been observed with a capsule occasionally. Therefore it resembles a pneumococcus. They prepared a vaccine fil-

trate and used it extensively in the treatment of these cases and have obtained very good results. When the lesions were low enough for irrigation they treated with silver nitrate, lunar caustic, mercurochrome and hot water. They also consider that an important adjunct to the treatment is the tincture of iodine by mouth.

The findings of this organism in distant foci and the production of acute lesions in the colon could account for the repeated exacerbations of the disease.

Buie (18) gave a very good description of the proctoscopic picture. "In the early stages the membrane is granular, bleeds easily, is diffusely edematous and hyperemic. This is followed by various gradations of ulceration. The small white spots noted in the hemorrhagic membrane were in fact small miliary abscesses which finally form superficial ulcers. Later on the membrane has a granular glazed appearance with only tufts of mucosa here and there. Small polypoid growths of mucous membrane are occasionally scattered throughout the region of ulceration. When healing occurs small pit-like scars are formed."

The culture of the diplococcus is really a very difficult matter. It grows readily in the brain broth culture but the isolation from other organisms requires a great deal of bacteriologic skill and patience.

The diagnosis of chronic ulcerative colitis cannot be made without the use of the proctoscope and the x-ray barium enema. In our experience a large number of the cases are limited to the rectum and pelvic colon and do not involve the rest of the colon before a later period. However, some of the X-ray plates show that isolated segments of the colon may be rarely involved.

I am sure that we have seen a large number of early cases involving the rectum and pelvic colon that have been completely arrested by means of local treatments. These local treatments have included mercurochrome in one per cent solution and the use of various powders applied by means of a special insufflation apparatus. Equal parts of calomel and bismuth subcarbonate is our favorite powder. After healing, characteristic pit like scars remain visible in various areas of the mucosa. In many areas the veins reappear and the membrane appears to be perfectly normal.

All the older proctologists describe a hemorrhagic proctosigmoiditis which is limited to the rectum and pelvic colon. It occurs in distinct attacks and is amenable to local treatment. I have verified this observation in a large number of cases and believe that the disease is a definite clinical entity. I have never seen a case that involved the whole colon. It usually clears up within two or three weeks' time under local treatment. It may be possible that it is a self limited disease and would disappear without any form of treatment.

Ileostomy has given a great deal of service in some of our cases but I would not advise the operation except as a last resort. Our best results have been secured by bed rest, high vitamin diet, as advocated by Dr. Larimore, and the use of mercurochrome in the rectum as well as the keratin coated tablets by mouth. I believe the best form of treatment to consist of keratin coated tablets of mercurochrome by mouth combined with local treatments of the lesions that are visible with the

sigmoidoscope. In some of the cases we have passed a small soft rubber catheter through the sigmoidoscopic tube and applied the powder by means of the insufflation apparatus. I would not advise dilatation of the colon in the late cases because of the friability of the gut. Blood transfusions are of great value in all cases of long duration.

Chronic catarrhal colitis may show a disappearance of the haustration in part of the colon particularly in the descending and pelvic colon, but it does not present the rigid gas-pipe like appearance of ulcerative colitis.

Patients with amoebic dysentery of long standing present an irritable colon with dilitations and contractures here and there without loss of haustration. Luetic lesions are usually limited to the rectum and the lower half of the colon and are most likely to form local stricture formations. Tuberculous ulceration of the colon does not result in the loss of haustration but forms isolated contractures and defects.

This study is based on thirty-two cases of chronic ulcerative colitis. The Bargen diplococcus was isolated in sixteen of them. The remaining sixteen cases were studied before the appearance of Bargen and Logan's paper. The vaccine filtrate was used in ten of the cases with somewhat disappointing results. I shall proceed with abstracts of several case histories illustrating our results in treatment.

Case 1.—Female, age 36. Came under observation in June 1922. She gave a history of chronic diarrhea of five years' duration. She consulted Dr. Bertnam Sippy three years ago and received various treatments including irrigation but continued to get worse. Finally in May 1921 the operation of ileostomy was performed under Dr. Sippy's direction. She was better for a time but the diarrhea persisted and she gradually became addicted to opium. She used the tincture, taking from ten to sixty drops daily. Has been an invalid since the operation, confined to bed a greater part of the time. The sigmoidoscope, one-half inch tube, could be introduced into the greatly contracted rectum. The mucosa was covered with blood and pus, thick, velvety and bleeds easily. Barium enema revealed the presence of high grade exudative colitis. Entire colon was dehaustrated, tubular and rigid. She has been kept under careful observation ever since. She was treated by irrigations of the colon through the ileostomy opening as well as by rectum. One per cent solution of mercurochrome gave her less pain then any other agent used. She continued to be a semi-invalid. Colon continued to contract until in the Spring of 1925 it was markedly smaller in caliber, the rectum contracted down so that it would receive only a three-eighths-inch tube. The mucosa of the rectum by this time had apparently entirely disappeared leaving only bloody scar tissue. X-ray taken at this time showed the cecum to be considerably dilated. She had a great deal of abdominal pain and had to use opium in larger doses. It was frequently found that the blood and pus discharge from the cecum would soil the ileostomy opening. Because of this fact, her continued invalidism and constant pain requiring the use of opium, colectomy was decided upon. The operation was performed by Dr. Vernon Mastin and because of the tremendous amount of fat in the mesentery, presented considerable technical difficulty. About six inches of the rectum was left. She made a very satisfactory recovery and has improved very much since that time. She is free from pain, never requires any sedative and expresses herself as being very comfortable, up and about, very active and cheerful. The ileostomy is easily managed and occasions no discomfort. Her diet consists of all well cooked foods plus fruit juices. Feces analysis shows soft, mushy stools, free from mucus, pus and blood with good digestion of the fats, starches and muscle fibers. It has a faint acid odor but is free from the odor characteristic of colonic feces.

Recent roentgenograms reveal that food remains in her alimentary tract fourteen

hours. The entire small intestine is better visualized than is usual. The barium remains in contact with the various segments a considerably longer time.

Description of the Resected Colon By Dr. Geo. Ives

The specimen in formaldehyd solution is a colon with small portion of ileum attached. It is 79 cm. in length from the tip of the cecum to the severed end.

The greatest diameter of the specimen is 4 mm. at the cecum, the smallest diameter 15 mm. at the severed end. The thickness of about 9 mm. The most prominent part of the specimen is a fatty submucosa which measures from 2 to 8 mm. in thickness with an average thickness of about 6 mm.

The colon is imbedded in a considerable amount of fat, the epiploica are large and pendulous, the fat shows a considerable amount of congestion and many small hemorrhages, possibly occasioned by the operative removal. The whole specimen has the appearance of being contracted. The wall is greatly thickened, the increased thickness being caused, for the most part or entirely, by a layer of fatty tissue between the circular muscle coat and the muscularis musosae. The thickness of the layer of fatty tissue varies from 2 mm. to 8 mm. and is continous throughout the specimen, both in the colon and in the ileum. To a slight extent the fatty layer continues into the appendix.

The mucosa is covered, except for a short distance at the severed end, by a thick friable fibrinohemorrhagic exudate, which is greatly cracked and fissured, and there can be found grossly, no evidence of a normal mucous lining. Small dried granular pieces 5 to 6 mm. in diameter have been detached from the inner surface in various places. These have the appearance of being dried blood clots. The muscle coats may be easily separated as may also the individual muscle bundles of the circular coat.

Microscopic Examination

The most prominent feature of each section is the fatty layer in the submucosa, which measures from 2 mm. to 8 mm. in thickness. This layer shows simply fatty tissue without any inflammatory reaction.

The surface of the mucosa throughout the specimen is covered with an acute hemorrhagic exudate. The mucous membrane is ulcerated in many places and where found is atrophic. The greatest amount of ulceration is in the transverse and descending colon. Beneath the mucous surface is a chronic inflammatory reaction consisting most of plasma cells. These cells extend down to the muscularis mucosae and make up most of the tissue of the mucosa, though a few polymorphonuclear leucocytes have infiltrated the tissue. This neutrophil reaction is most marked in the descending colon and is entirely absent in the ileum and cecum. Throughout the mucosa are found epitheloid cells, many of them showing mitotic figures.

The gross and microscopic picture is that of an acute ulcerative colitis superimposed on a chronic inflammation of the mucous membrane, plus the marked fatty infiltration of the submucosa.

This report of Dr. Ives differs from other descriptions that I have seen in the literature. I believe no one else has called attention to the tremendous amount of fatty tissue in the submucosa. There does not appear to be any fibrosis or connective tissue increase.

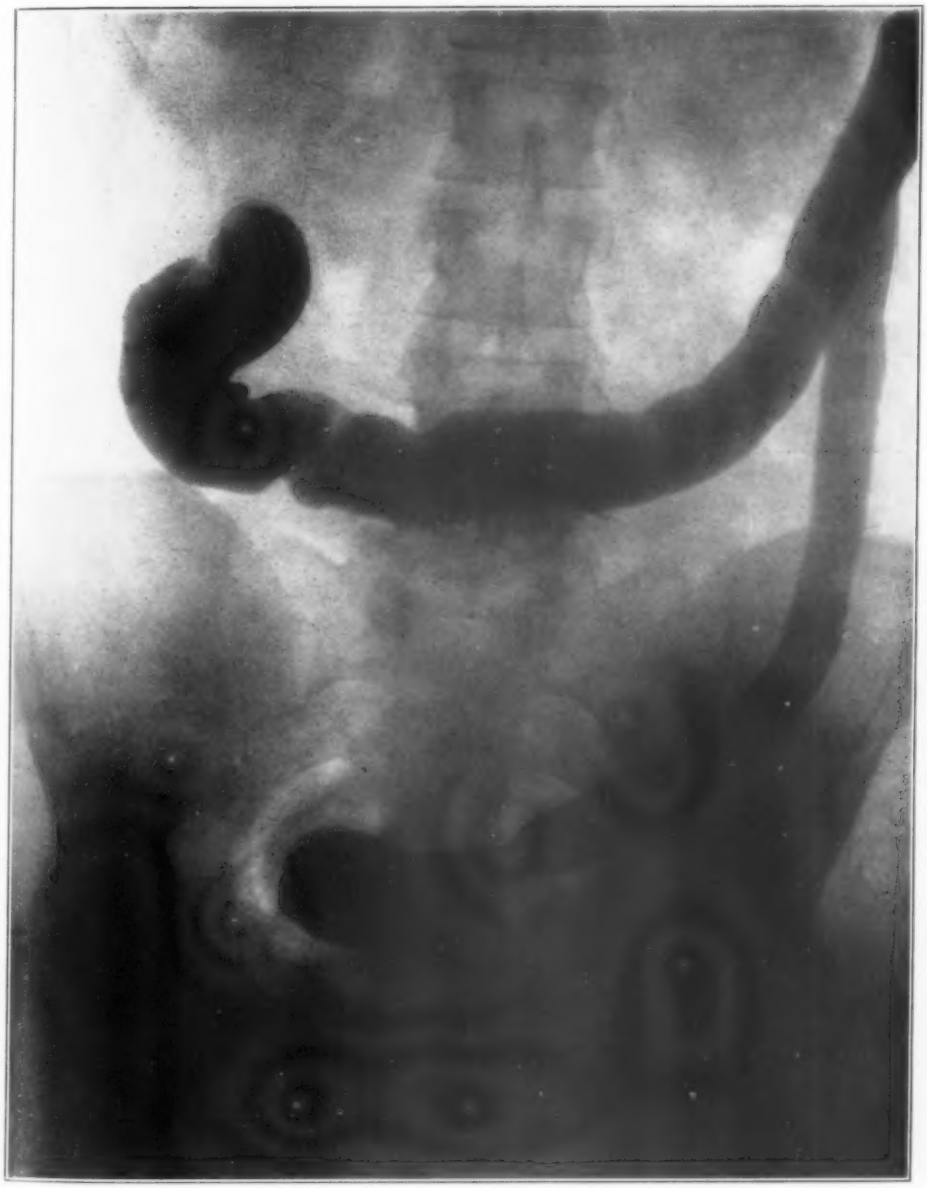

FIG. 1. Case I. This film was taken when patient first came under treatment (June 1922).

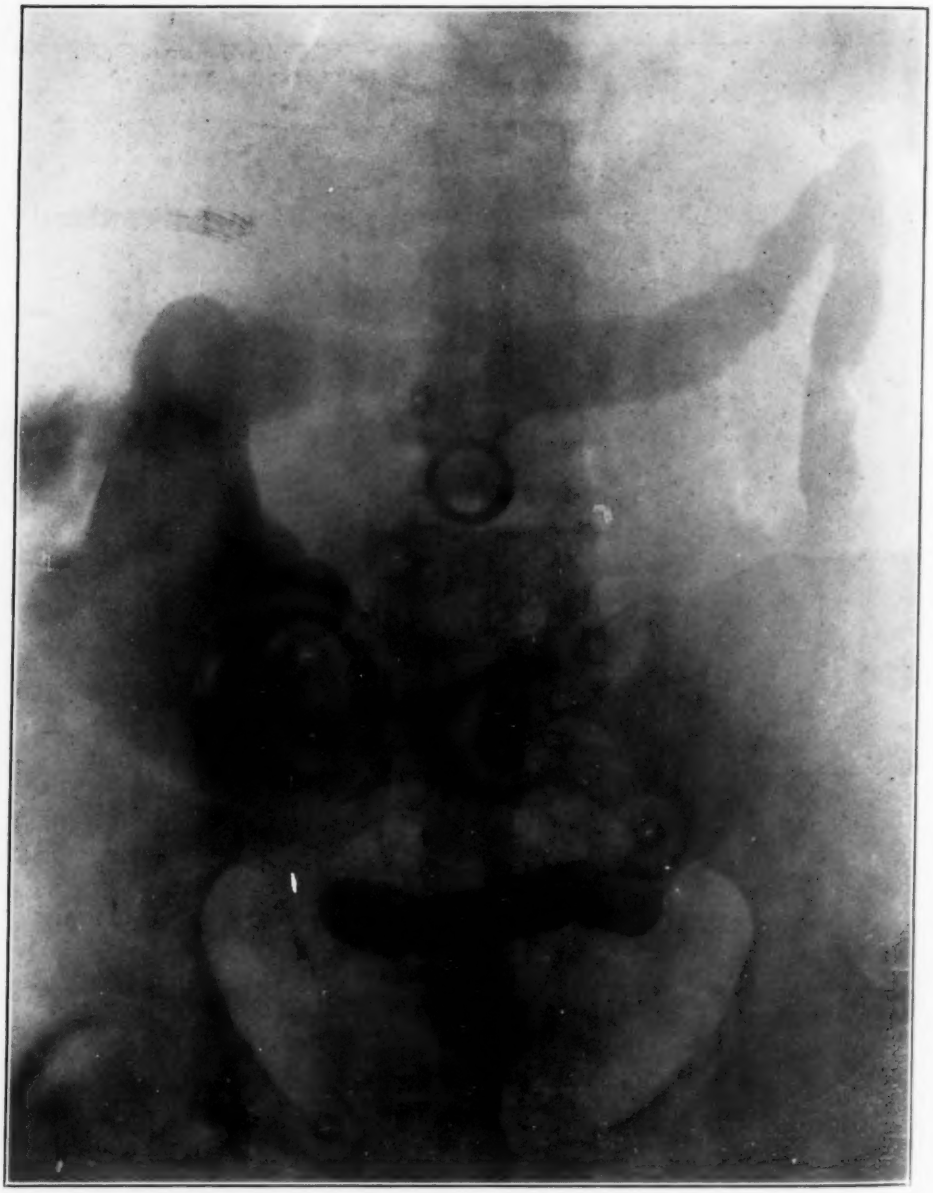

FIG. 2. Case I. This film was taken a short time before operation. Shows very much dilated cecum which gave a great deal of pain and which was in reality a large abscess cavity.

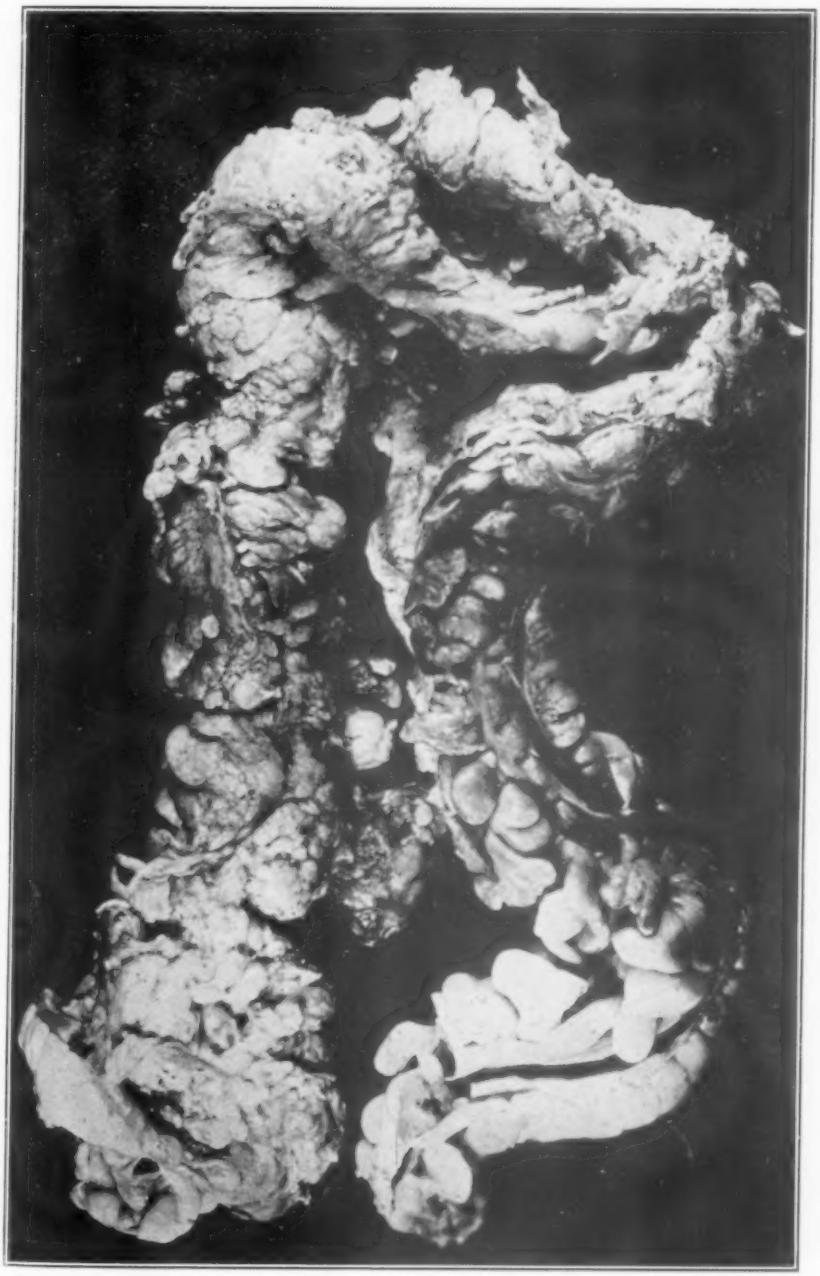

FIG. 3. Case I. The complete specimen showing the shrunken contracted condition of the colon and fatty epiploica.

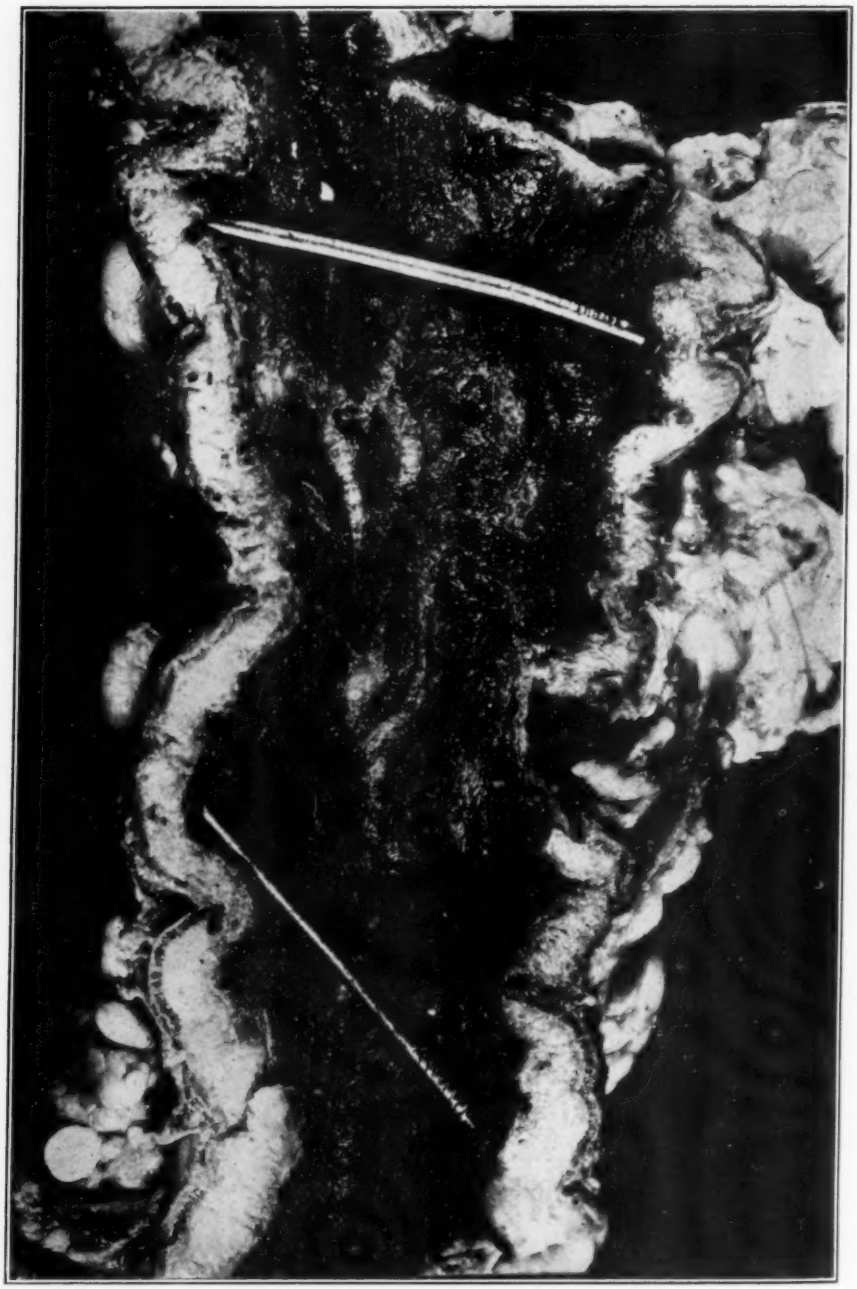

FIG. 4. Case I. Transverse colon spread apart to show the hemorrhagic condition of the mucosa and the thick fatty layer between the mucosa and the muscular wall.

FIG. 5. Case 1. Section through wall showing the relative thickness of the mucosa, fatty submucosa and the muscular coat.

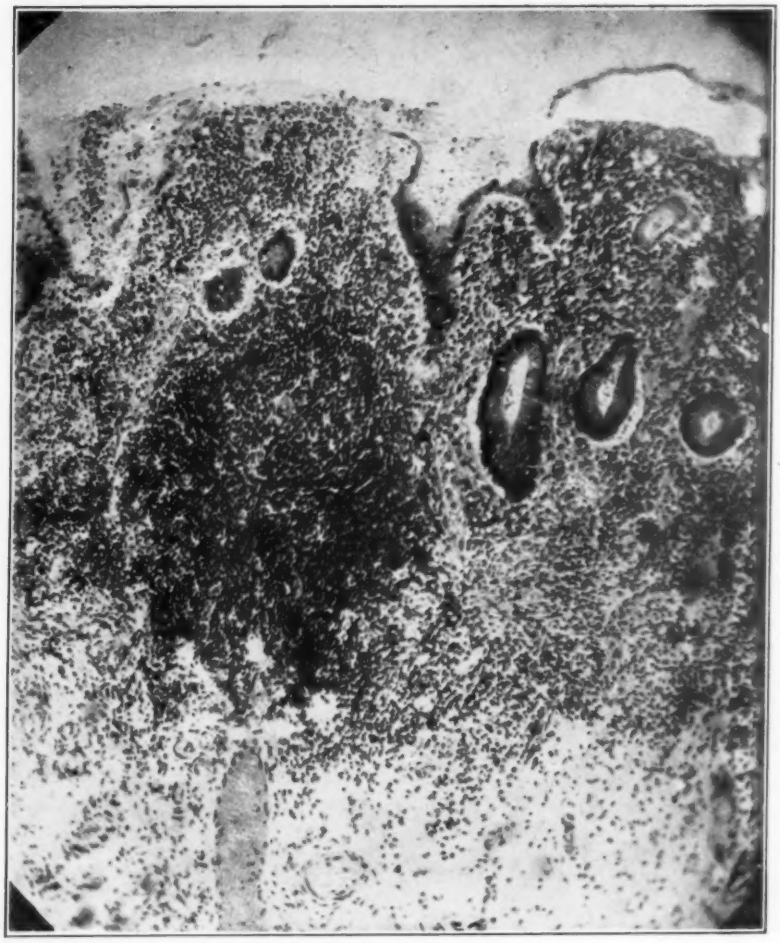

FIG. 6. Case I. Low power of the mucosa showing atrophy and ulceration of glandular epithelium, the acute exudate on the surface and the plasma cell reaction in the mucosa.

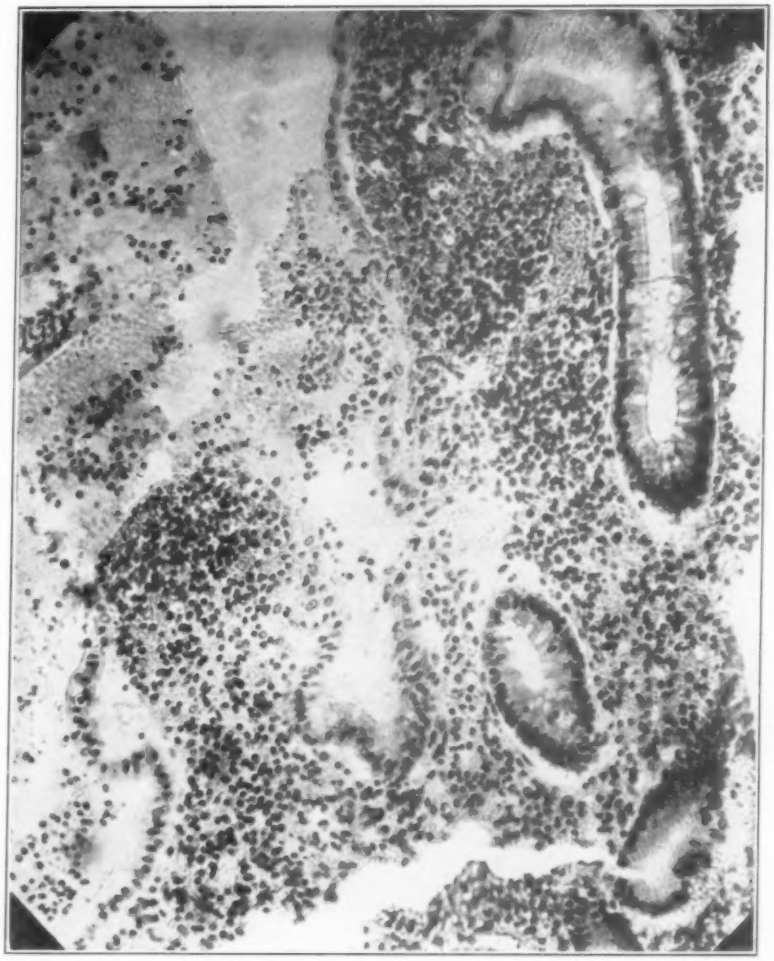

FIG. 7. Case I. High power showing acute inflammatory reaction on the surface and the plasma reaction in the mucosa.

Case II.—Female, age 26. Came under observation Feb. 19, 1920. Gave a history of diarrhea of one year's duration. Diagnosis of exudative colitis was made together with severe form of secondary anemia. Spleen was not enlarged. Normal weight 125, present weight 87½ pounds. General condition very poor. March 5, 1920 Dr. Harvey G. Mudd operated upon her. Cecum found to be very much thickened and involved in the inflammatory process. Appendix also much thickened. Appendix removed. Ileum then brought up and a portion about four or five inches from the ileo-cecal valve was fastened to the wound. She improved very much in her general condition after the ileostomy until the latter part of the following August when she had a very acute attack with extension of the process into the ileum. She died of acute enteritis.

Case III.—Female, age 25. Came under observation Feb. 8, 1926. Had had diarrhea for nearly a year, recently blood and pus appeared in the feces. Her normal weight was 137 pounds, when she came under observation 116¾ pounds. Her spleen was definitely enlarged. Blood test showed severe secondary anemia. X-ray showed that the colon was involved to the splenic flexure. Procto-sigmoidoscopy revealed characteristic picture of ulcerative colitis. Entire bowel atonic in character. She was put on mercurochrome and high vitamin diet. Had very little appetite and soon developed fever. Sent to St. Luke's Hospital. She was greatly prostrated, ran very high temperature curve, ranging between 102-105. Mercurochrome had to be discontinued because of her inability to retain it. Was delirious and very ill. Finally determined on ileostomy. Dr. Vernon Mastin performed the operation March 19, 1926. Terminal ileum was definitely involved in the inflammatory process. The ileostomy opening was made six inches from the distal end of the ileum. Patient succumbed seven days later from peritonitis. This was a severe acute fulminating case.

Case IV.—Male, age 56. Came under observation April 25, 1921. Colitis seven years' duration. Often had thirty movements daily. X-ray examination showed the loss of haustra in the colon characteristic of chronic ulcerative colitis. In May 1921 the operation of ileostomy was performed. The ileum was severed from the colon and the distal end was closed near the ileo-coecal valve. Proximal end brought through the muscle and fixed. He gained in weight and made considerable improvement. Did not have much trouble with the ileostomy opening. In June 1924 X-ray films showed colon to be extremely narrow excepting the cecum which was dilated. He was having at this time considerable pain in this region because of insufficient drainage. He continued to grow weaker and finally succumbed in June 1926 to perforation and peritonitis. Colectomy might have saved this patient but his general condition did not warrant it. The double opening in the ileum is to be preferred because drainage of the cecum is necessary.

Case V. Female, age 32. Came under observation Nov. 5, 1923. Gave history of colitis of twelve years' duration. In 1914 appendicostomy was done which appeared to make her much worse. In 1916 ileostomy was performed under Dr. Bertram Sippy's direction. The rectum was so contracted that it would not retain the barium enema. Barium was injected through the ileostomy opening and part of the colon visualized. Her general condition was very satisfactory. She was not suffering any pain and the ileostomy opening was functioning very satisfactorily. Feces analysis showed that the ileum was not involved in the inflammatory process. She consulted me in regard to colectomy. I advised against it and furthermore advised her not to attempt any irrigation of the colon but to let it alone.

Case VI.—Male, age 17. Came under observation May 13, 1925. X-ray films and proctosigmoidoscopy showed picture of chronic ulcerative colitis. Two years duration. Recent exacerbation of the condition with blood and pus mixed with the feces. Treatment consisted of high vitamin diet, insufflations of Scarlet R and calomel and bismuth powder and mercurochrome keratin coated tablets. In November Lugol's solution was

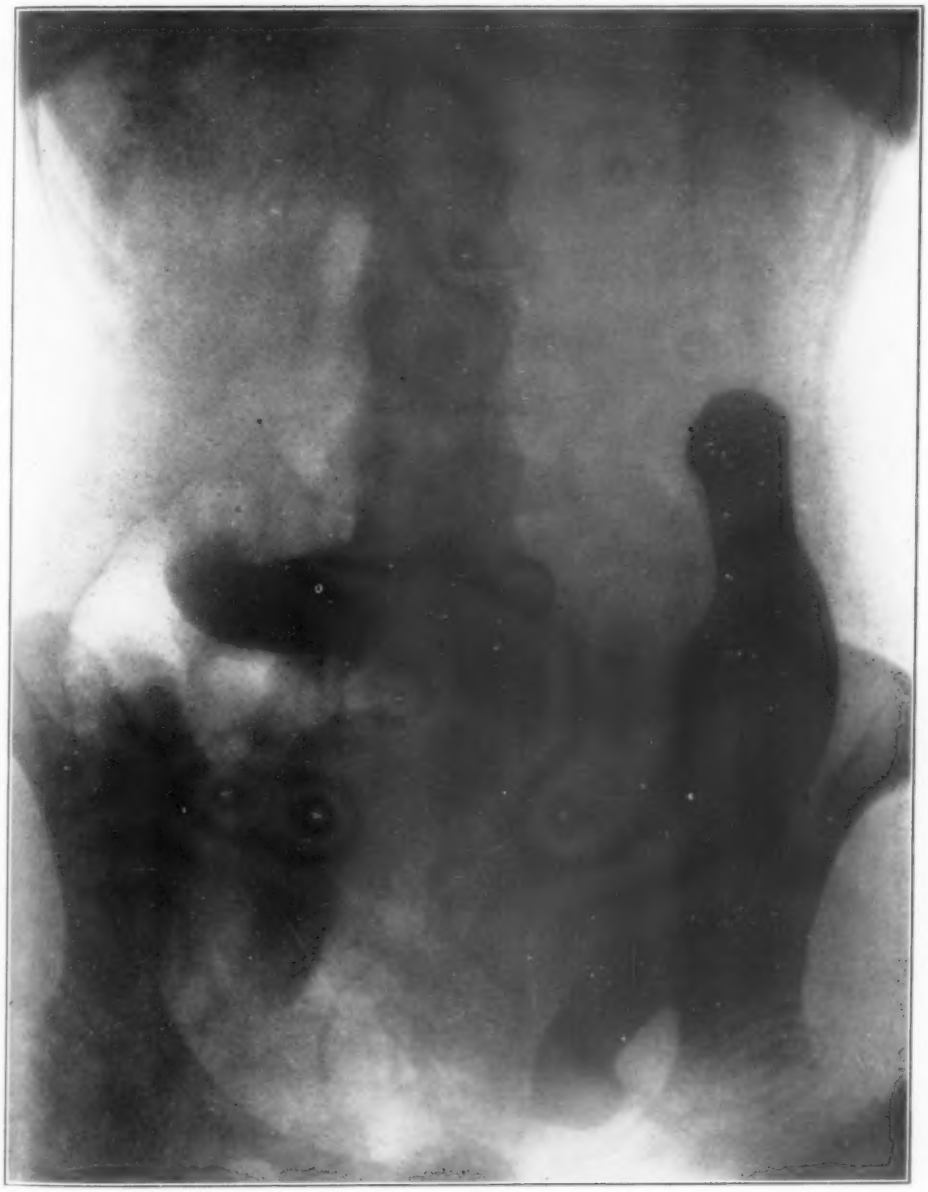

FIG. 8. Case II. Patient succumbed to enteritis six months after ileostomy.

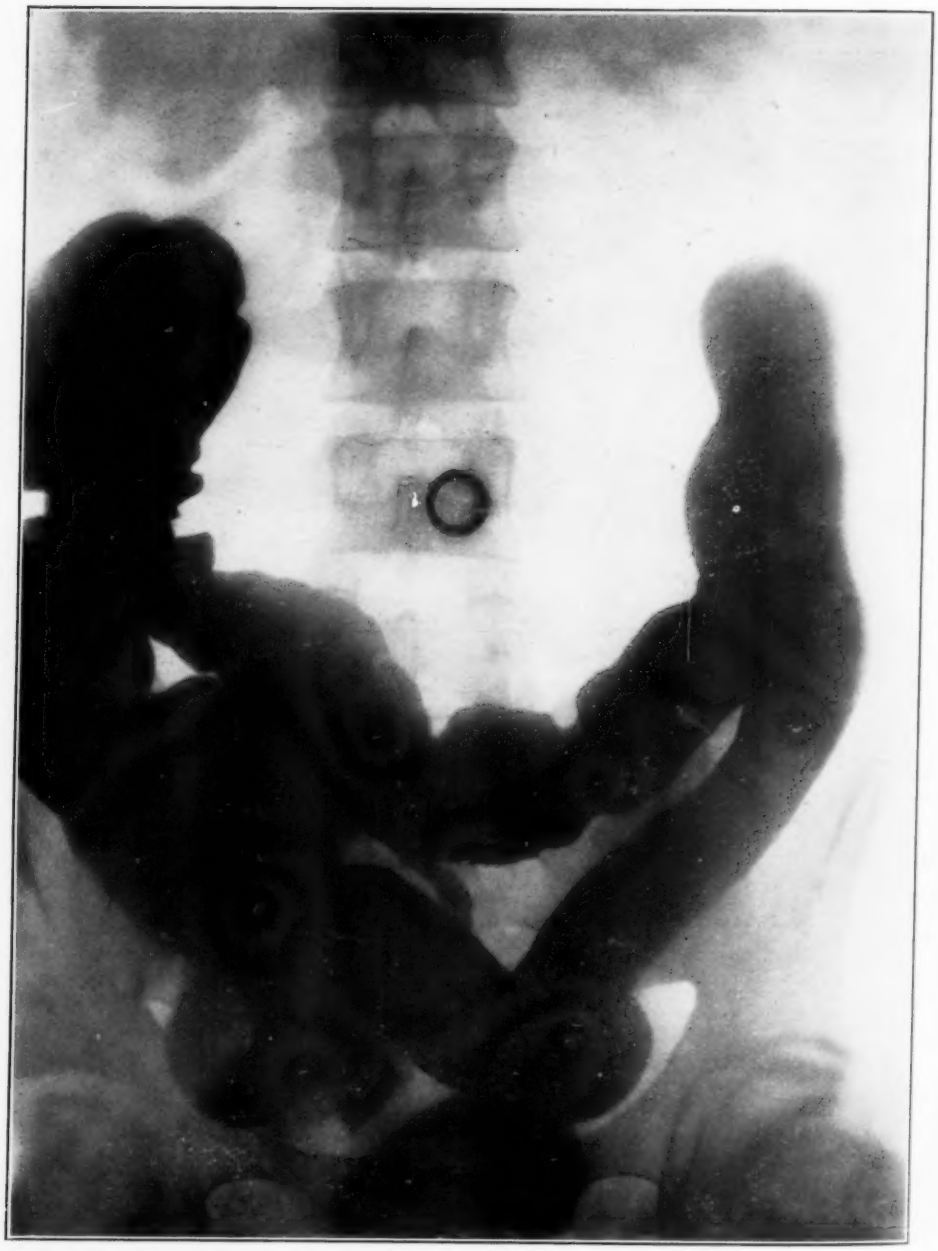

FIG. 9. Case III. A fatal case of the fulminating type of ulcerative colitis.

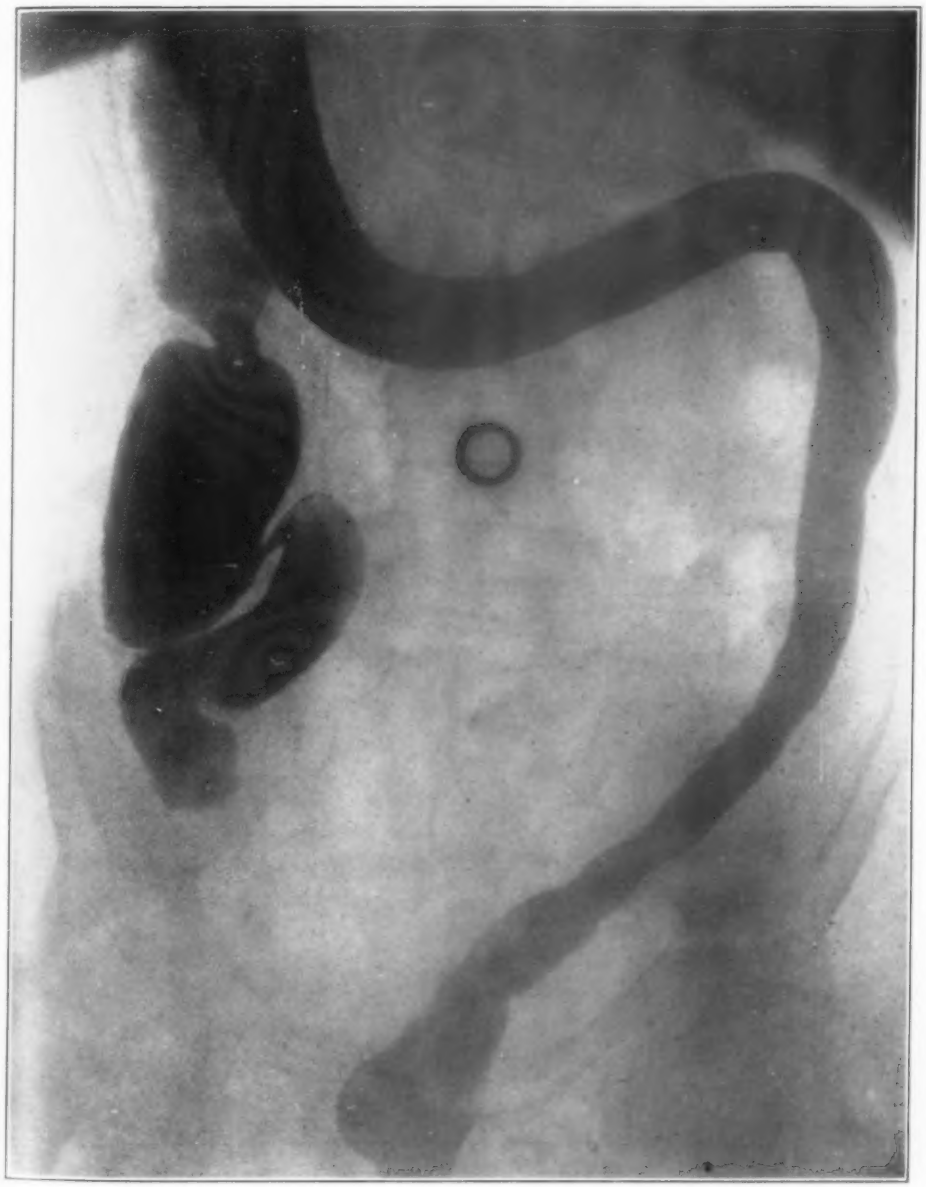

FIG. 10. Case IV. This film shows the condition when patient came under observation in April 1921.

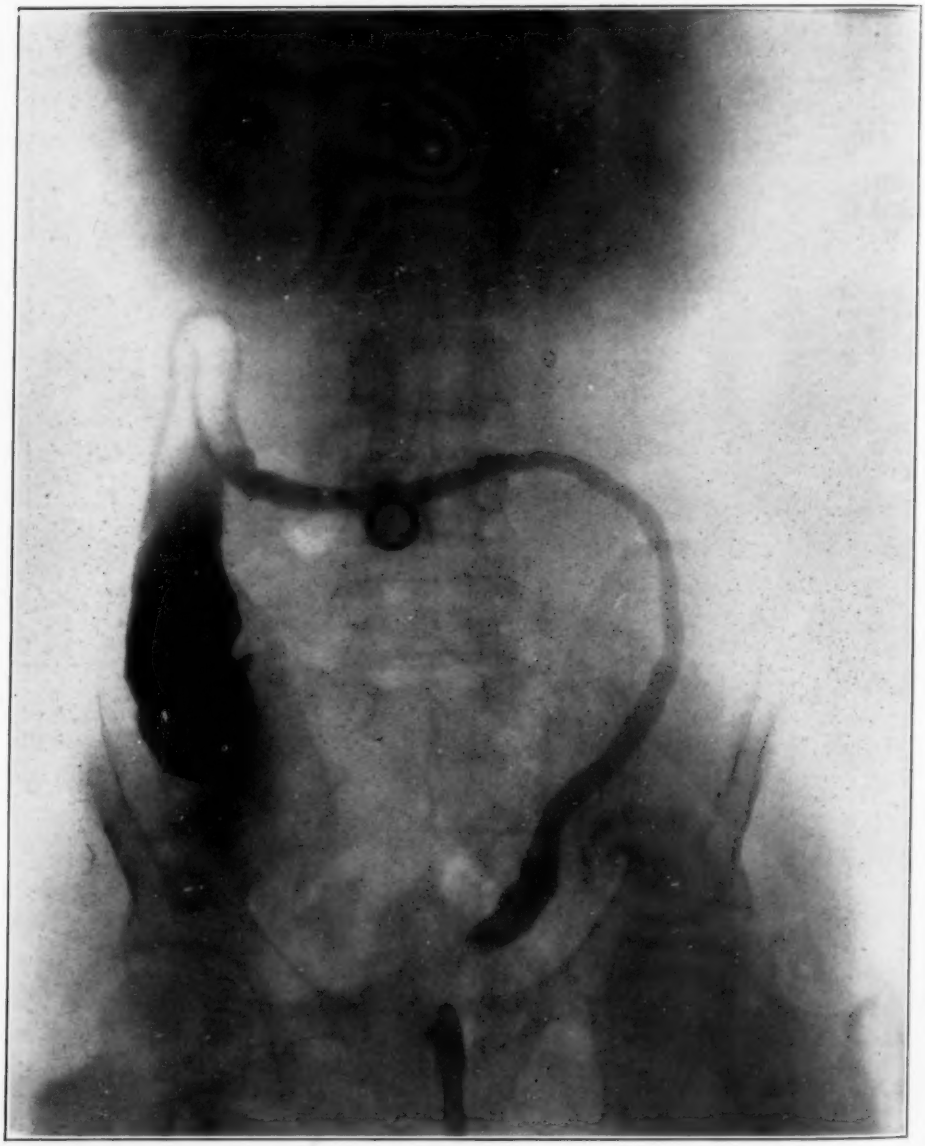

FIG. 11. Case IV. This film was taken three years after the operation of ileostomy. Note the narrowing of the entire colon except cecum which is dilated.

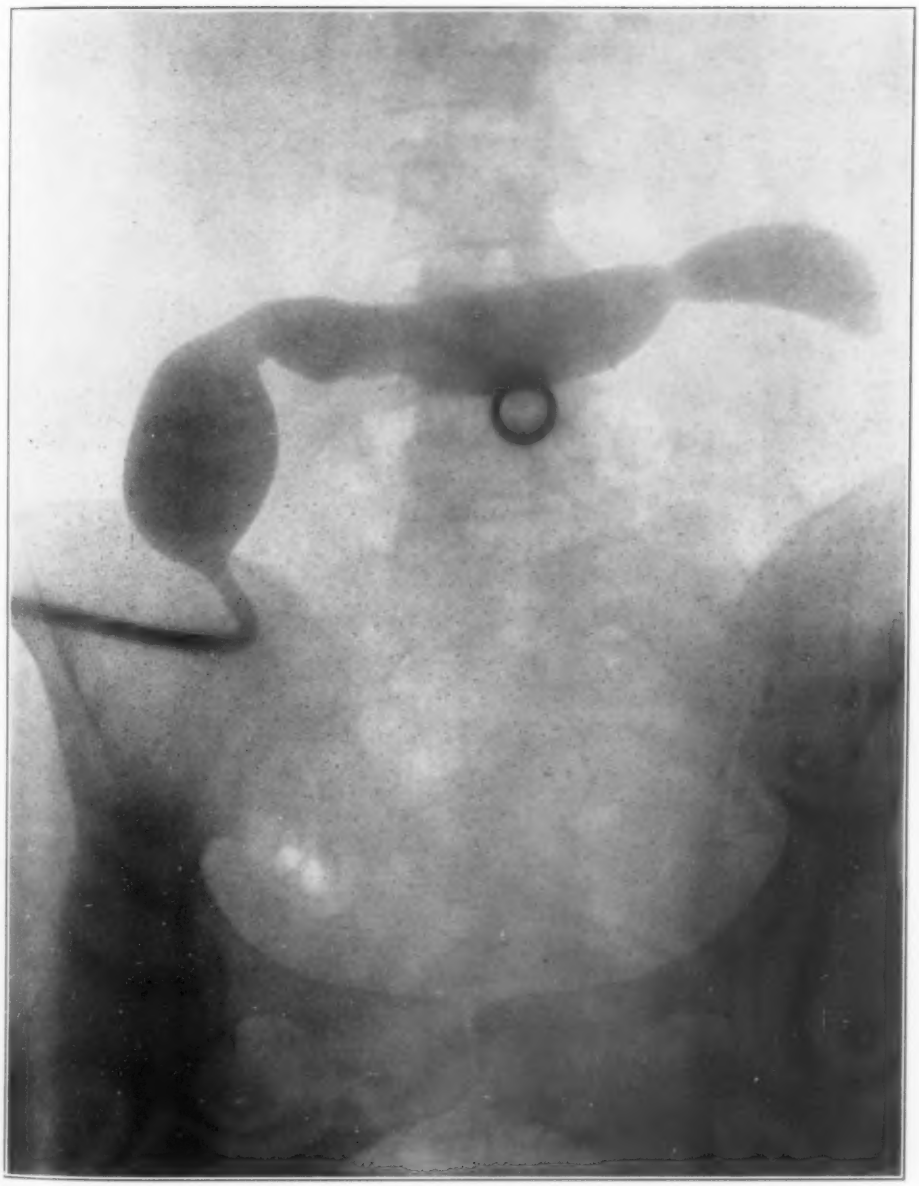

FIG. 12. Case V. The distal colon and rectum was obliterated seven years after ileostomy.

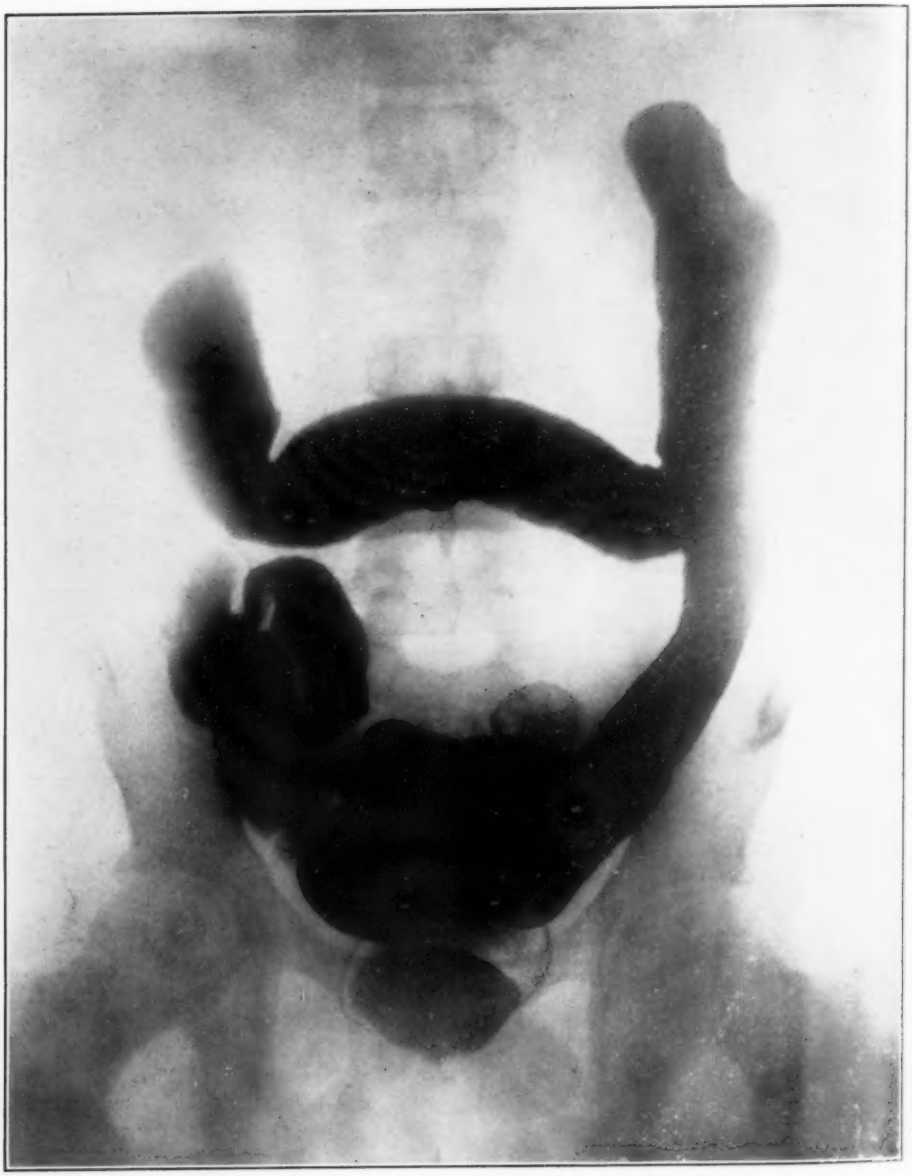

FIG. 13. Case VI. (May 15, 1925) Showing chronic ulcerative colitis involving entire colon.

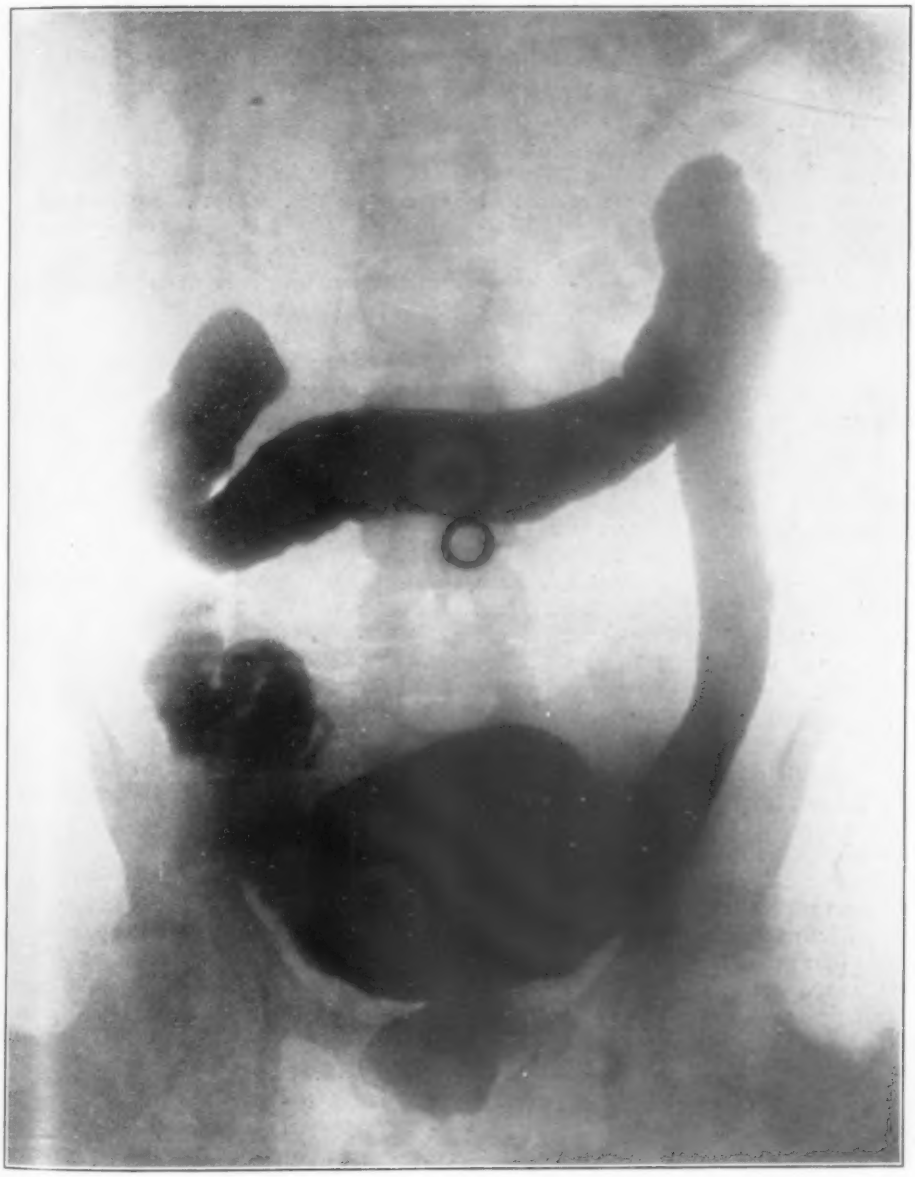

FIG. 14. Case VI. (July 20, 1926) Possibly slight improvement as shown by the wider transverse colon and possibly some increase in calibre of the descending colon.

given. In December the vaccine filtrate was given. The vaccine therapy appeared to be helpful in this case. He is now clinically well but the colon shows little if any change.

Case VII. Male, age 22. Came under our care in Oct. 1925. Prior to that time was under Dr. L. E. Printy's care from May 14, 1925. Gave a history of diarrhea for two years' duration. His treatment in our office consisted of one percent solution of mercurochrome given through sigmoidoscopic tube. He made but little improvement. Dr. Larimore began treatment solely by his high vitamin and high calorie diet. He made a remarkable recovery.

Case VIII. Female, age 51. Came under observation Jan. 1923. Gave history of rectal trouble of two years' duration. Attacks of diarrhea. Stools contained blood and mucus. Has had no hemorrhoids. X-ray showed exudative colitis, dehaustration and narrowing of the descending iliac and pelvic colons. Sigmoidoscopy revealed a mucous membrane characteristic of ulcerative colitis. She responded very well to local treatment consisting of calomel and bismuth powder. The condition appeared to be normal at the end of about two months' treatment. She had a recurrence in Jan. 1924 but responded well to local treatment. In July 1925 she came in for general digestive disturbance and the membrane was found to be characteristic of a healed ulcerative colitis. She remained well until July 1926 when she had a more severe attack than the previous one. She was given the mercurochrome tablets by mouth but they seemed to aggravate the diarrhea and they were discontinued. The mercurochrome given by rectum was not well borne. Therefore, she was given local treatments with the powder insufflator using the bismuth and calomel powder. At the present time her membrane shows characteristic pitting of healed ulcerative colitis and is free from blood, pus and mucus. She is feeling well, is on general diet omitting raw foods excepting orange juice. X-ray taken Sept. 1926 was exactly the same as the plate taken by Dr. Mills in 1923. This plate was unfortunately broken. This is a true case of chronic ulcerative colitis involving a portion of the descending, iliac and pelvic colons and rectum. Culture taken directly from the mucosa by Bargen's method. The diplococcus appeared in the brain broth but could not be isolated. Therefore, the vaccine was not used.

Case IX. Male, age 37. Came under observation Feb. 24, 1926. Bloody diarrhea of three months' duration. Severe pains, loss of twenty pounds in weight. Proctosigmoidoscopy shows characteristic membrane in the acute stage of colitis. X-ray films reveal that the process stopped sharply at the splenic flexure. He was given local treatments of mercurochrome and powder, high vitamin diet, mercurochrome keratin coated tablets by mouth, milk injections by Dr. Finnegan. The culture in this case developed the Gram-positive diplococcus which was isolated and pure culture obtained. Vaccine filtrate prepared. The vaccine injections appeared to do him a great deal of good. His rectum continued to give him a great deal of pain and it was found that a polypoid growth had developed at the internal sphincter. The growth had the appearance of carcinoma. On November 15, 1926 growth was removed by diathermy. He is at present gaining in weight and strength, has from one to two formed bowel movements daily. Membrane now presents appearance of a healed ulcerative colitis.

Case X. Female, age 16. Referred by Dr. A. E. Taussig. Came under our observation Sept. 13, 1926. Had been under treatment at Jewish Hospital. High vitamin diet and local irrigations. I saw her in consultation in June 1926. At that time mercurochrome keratin coated tablets, grains one and one-half, were given and three ounces of one percent solution of mercurochrome was introduced per rectum once daily. July 25, 1926 she developed mild symptoms of ptyalism and mercurochrome was discontinued. She was given a blood transfusion and left the hospital August 10th. She was given local treatments at my office consisting of insufflation of bis-

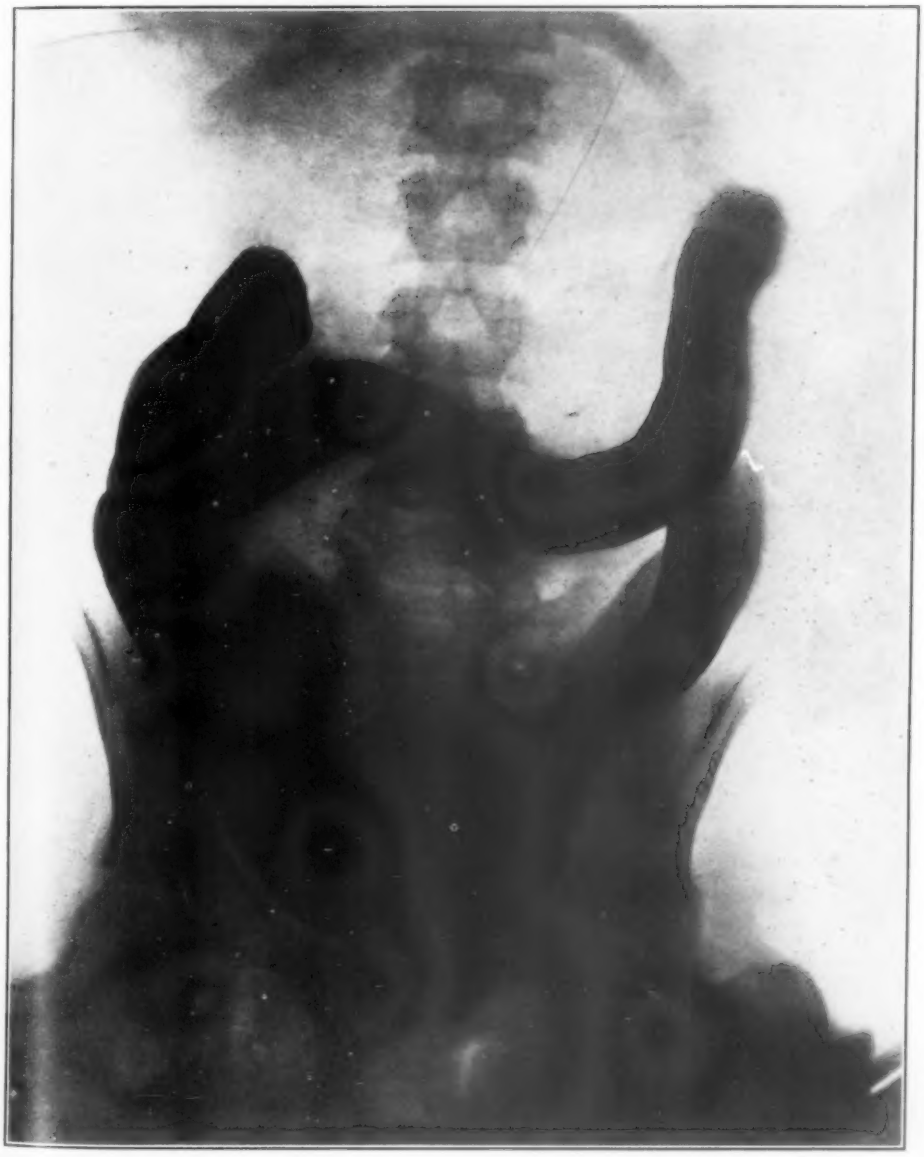

FIG. 15. Case VII. (July 28, 1925) Shows characteristic picture of chronic ulcerative colitis involving entire colon.

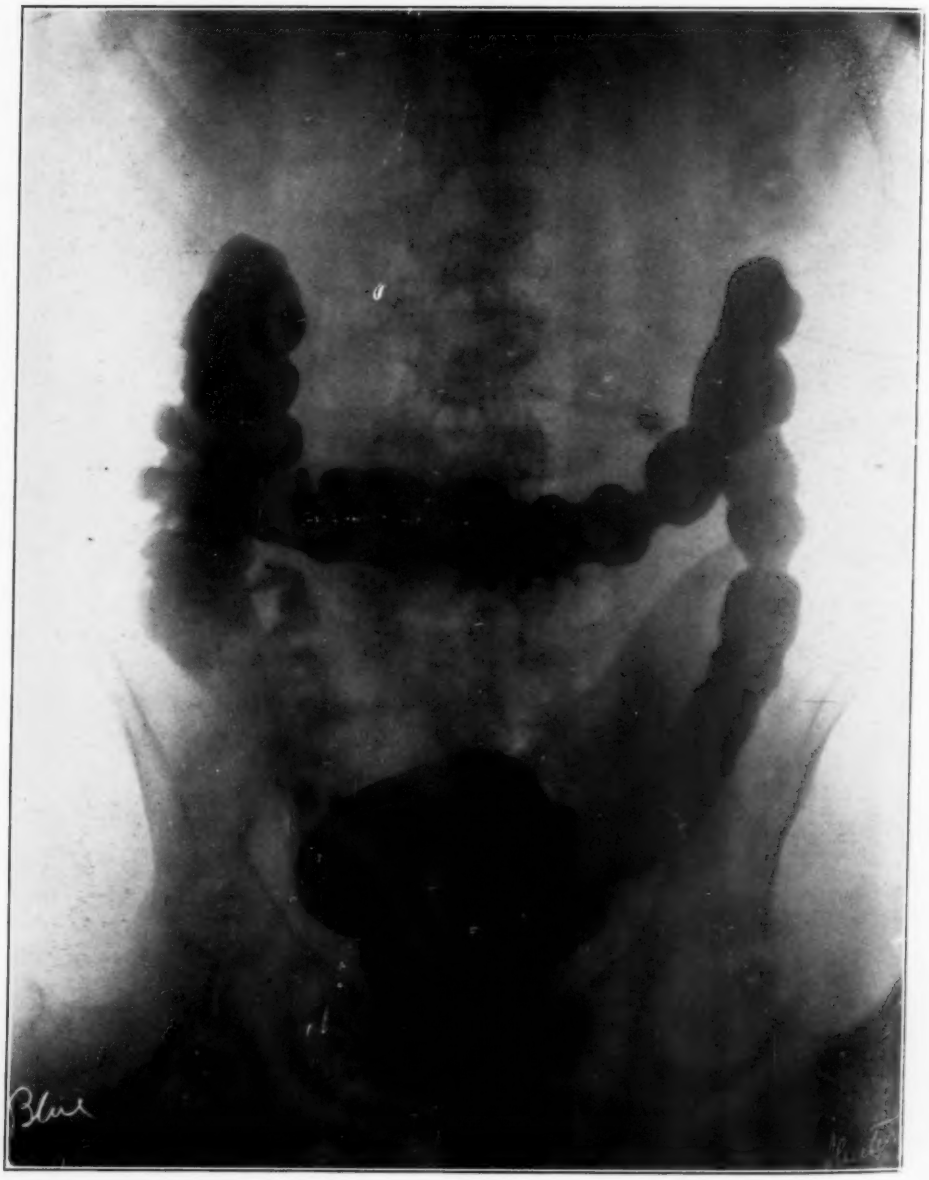

FIG. 16. Case VII. Shows marked improvement after high vitamin diet (May 11, 1926).

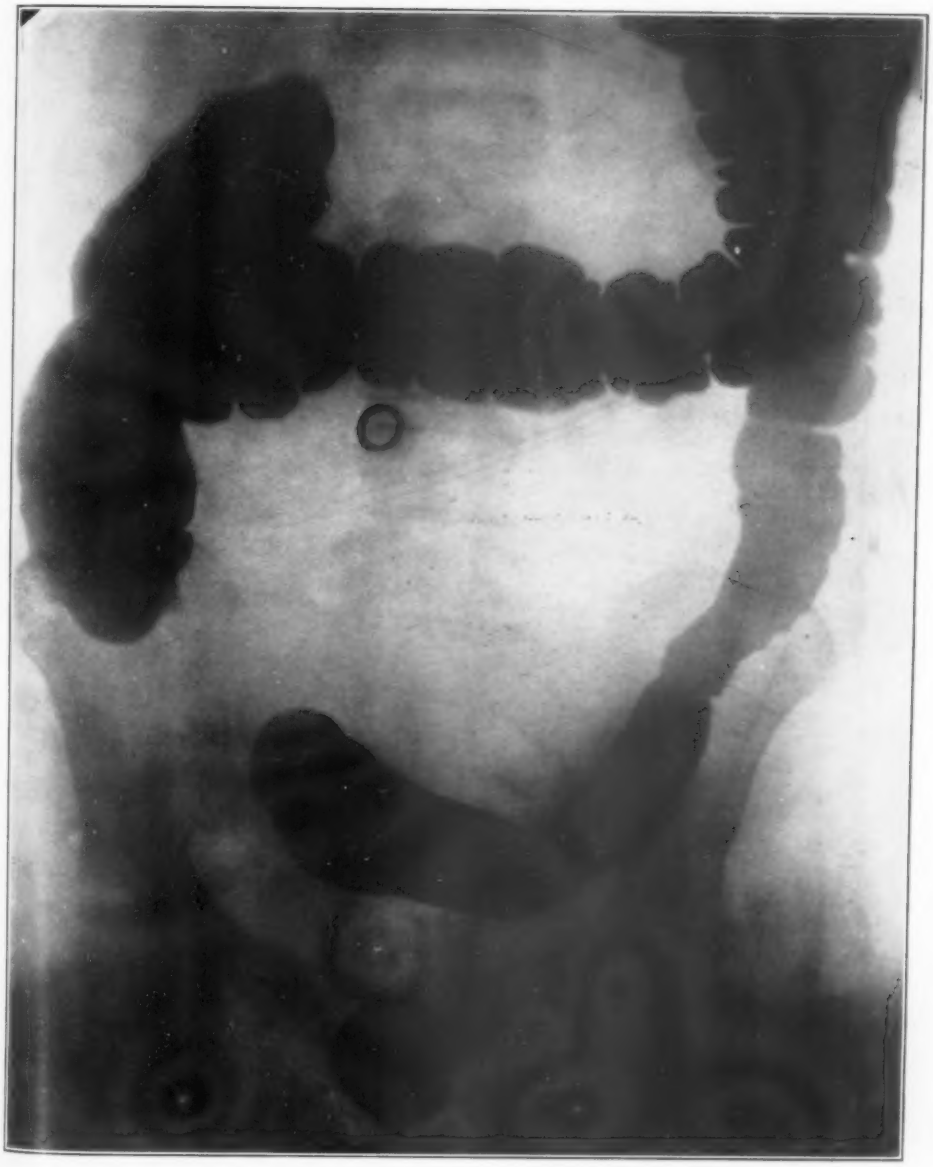

FIG. 17. Case VIII. A case of ulcerative colitis limited to the rectum and pelvic colon.

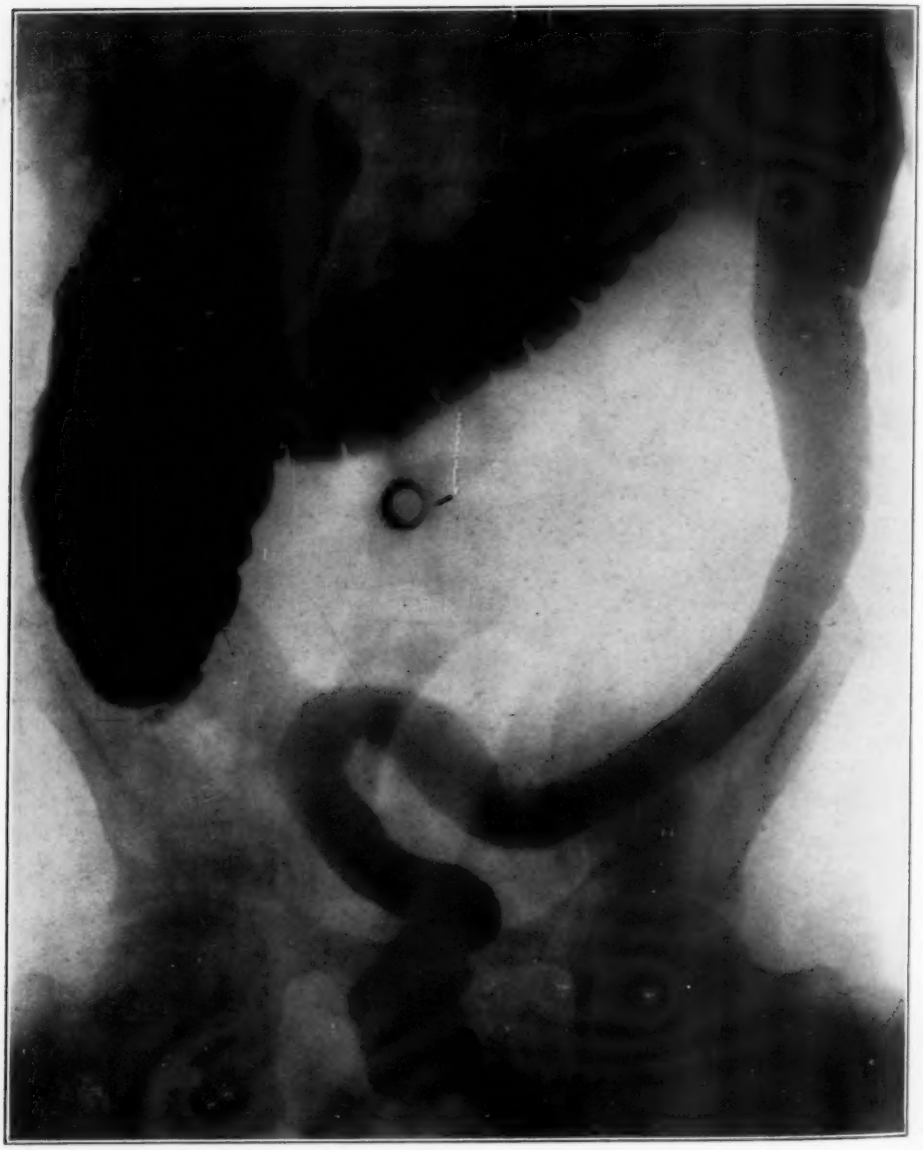

FIG. 18. Case IX. Colon was found to be involved from the anus to the splenic flexure. All the films were essentially the same as this one.

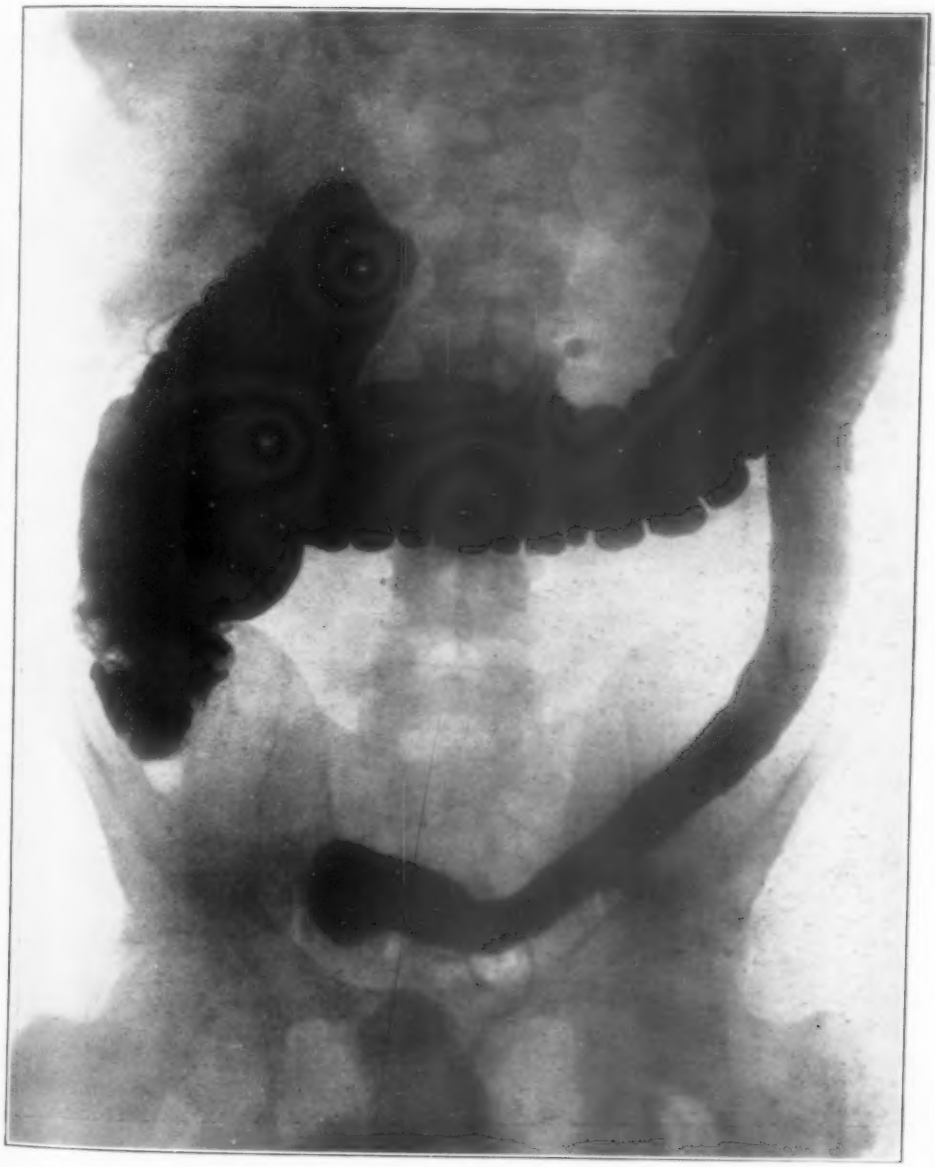

FIG. 19. Case IX. Just above the transverse flexure is one of the mercurochrome tablets not yet dissolved in the stomach.

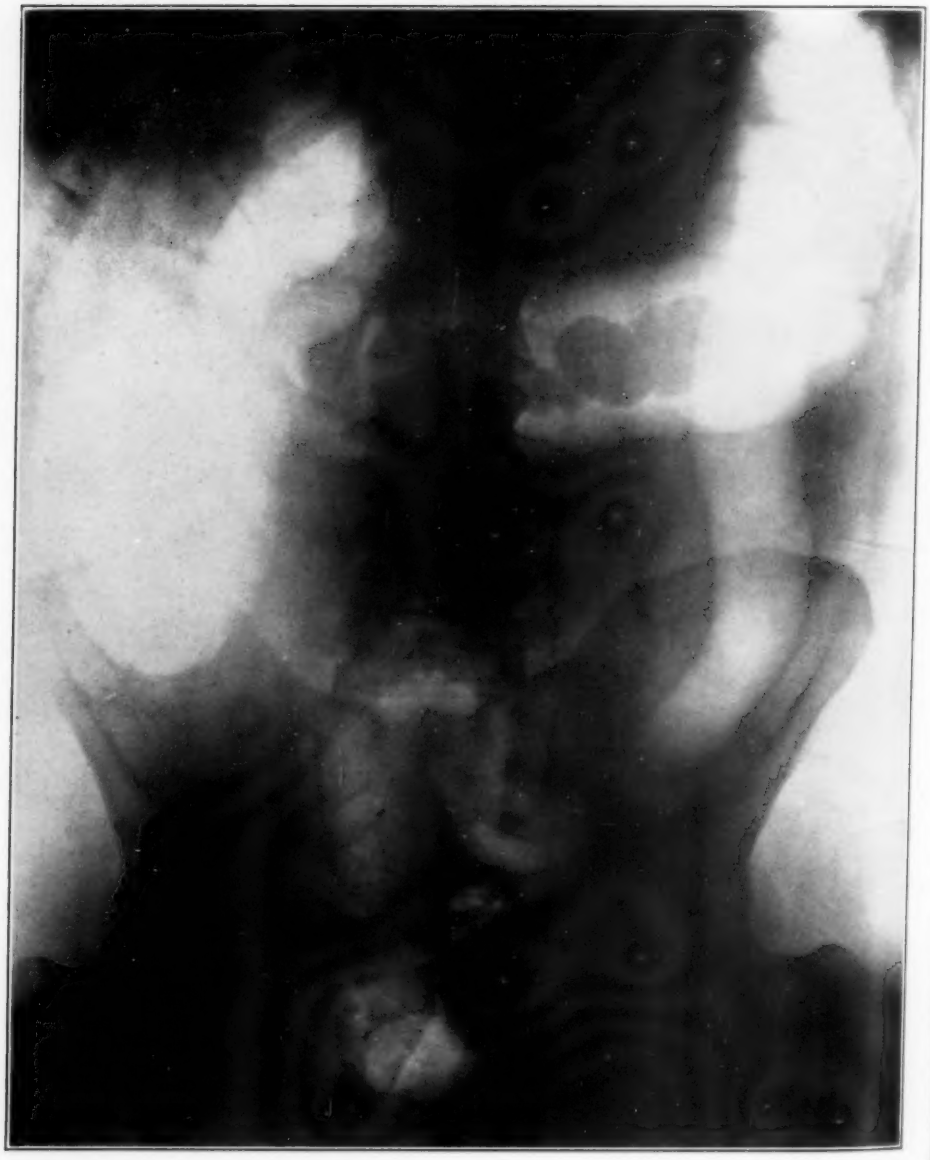

FIG. 20. Case IX. Colonic insufflation. Note that the haustra are visible in the transverse colon, the narrow descending and iliac colon showing the absence of haustration characteristic of ulcerative colitis. Note here the collection of fecal matter in the transverse colon. The patient had not been taking bismuth. This case illustrates what Dr. Mills designated as peripheral colonic motility.

muth and calomel powder. Bargen's diplococcus was finally isolated and vaccine filtrate prepared. In November she had adenitis involving the posterior cervical glands. High temperature 103. Again sent to Jewish Hospital. Her glandular enlargement subsided within a week's time. She was given another blood transfusion and discharged from the hospital. There was no exacerbation of the colitis during this acute attack. Feces analysis taken recently showed a few clumps of mucus, no blood or pus cells. At the present time she appears to be perfectly well. The membrane shows characteristic pitting of healed ulcerative colitis. She has continued to gain in weight and strength.

Case XI. Female, age 18. Entered Jewish Hospital Nov. 10, 1926. Had suffered from bloody diarrhea of three weeks' duration. Past history essentially negative. Examination showed poorly nourished, sallow complexion. Tonsils out, heart and lungs negative, tenderness in left lower quadrant. Sigmoidoscopy showed typical picture of ulcerative colitis. Temperature runs from 101-103. Pulse from 110-140. Gastric analysis showed achylorhydria. Stool examination shows watery stools with considerable blood, pus and mucus. Urinalysis showed trace of albumin, otherwise negative. Blood picture is that of secondary anemia. Differential count normal. She was given blood transfusion, Bargen's vaccine filtrate and high calorie, high vitamin fiber free diet. She made no response to treatment. Finally sigmoidoscopy revealed the presence of numerous polyps in upper rectum and pelvic colon. X-ray plate revealed a polypoid condition. Ileostomy has been done recently and she is now showing rapid gain in weight and strength. Colectomy will be advised later because the proctosigmoidoscope reveals several polypi that are already ulcerated and probably undergoing malignant degeneration. (Ileostomy was done by Dr. S. E. Newman).

Case XII. (Courtesy of Dr. Larimore) Female, age 16. Entered Barnes Hospital Sept. 3, 1925. Onset of present illness Feb. 1925. Abdominal discomfort which gradually became pain. Sigmoidoscopy revealed the presence of a normal rectum and pelvic colon. Gastro-intestinal X-ray examination showed contractural narrowing of the ascending and the transverse colons (more especially the proximal portion), and abnormal rugae markings of the descending colon. The changes in the more distal abnormal portions are probably of the same process less advanced, i.e. smaller, more sessile polyps. Diagnosis: Polyposis of the colon, benign. Operation by Dr. Evarts A. Graham. The transverse colon exhibited several small white nodules on the surface and the cecum showed gross evidence of disease. The sigmoid was practically normal. The colon was resected, including three inches of the ileum down to the middle portion of the sigmoid. The ileum was sutured to the sigmoid, over a rubber tube inserted through the rectum from below. Tube pulled down, inverting ileum into the sigmoid according to the Balfour method. The patient withstood the operation well. Pathological report, "The material consisted of a cecum, the ascending colon, transverse colon, and most of the descending colon, a meter in length. The cecum was normal and beginning at a point 4 cm. above the ileo-cecal valve were three irregular long ulcers extending to within 7 cm. from the end of the specimen. Each of these seemed to follow a muscle band. In addition, scattered here and there, over the mucosa were smaller, irregular ulcers. Lymph nodes seen on the outside of the intestine were enlarged but showed no gross change beyond a chronic inflammation. The mucosa itself showed the folds greatly exaggerated and numerous small polypi were seen. Microscopic Report: The sections showed large intestine in which there was thickening in most places of the submucosa and marked infiltration of all the layers with round cells. The mucosa especially was filled with these cells. In addition, there was increased vascularity and all the vessels were filled with blood. In places the epithelium was missing and was replaced by areas of necrotic cells and polymophonuclears and round cells. No evidence of any specific process was seen. There was some increase in the size of

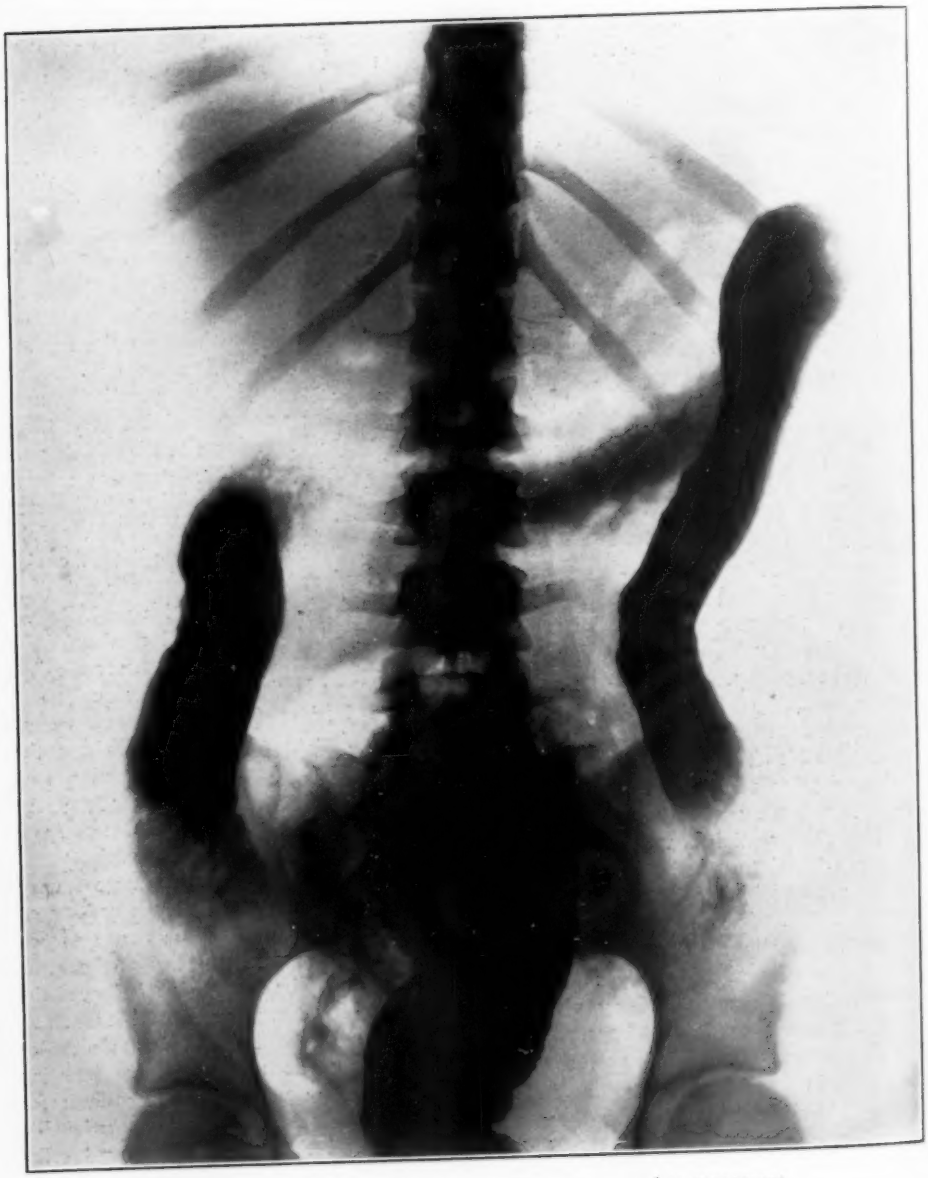

FIG. 21. Case X. When patient first came under treatment.

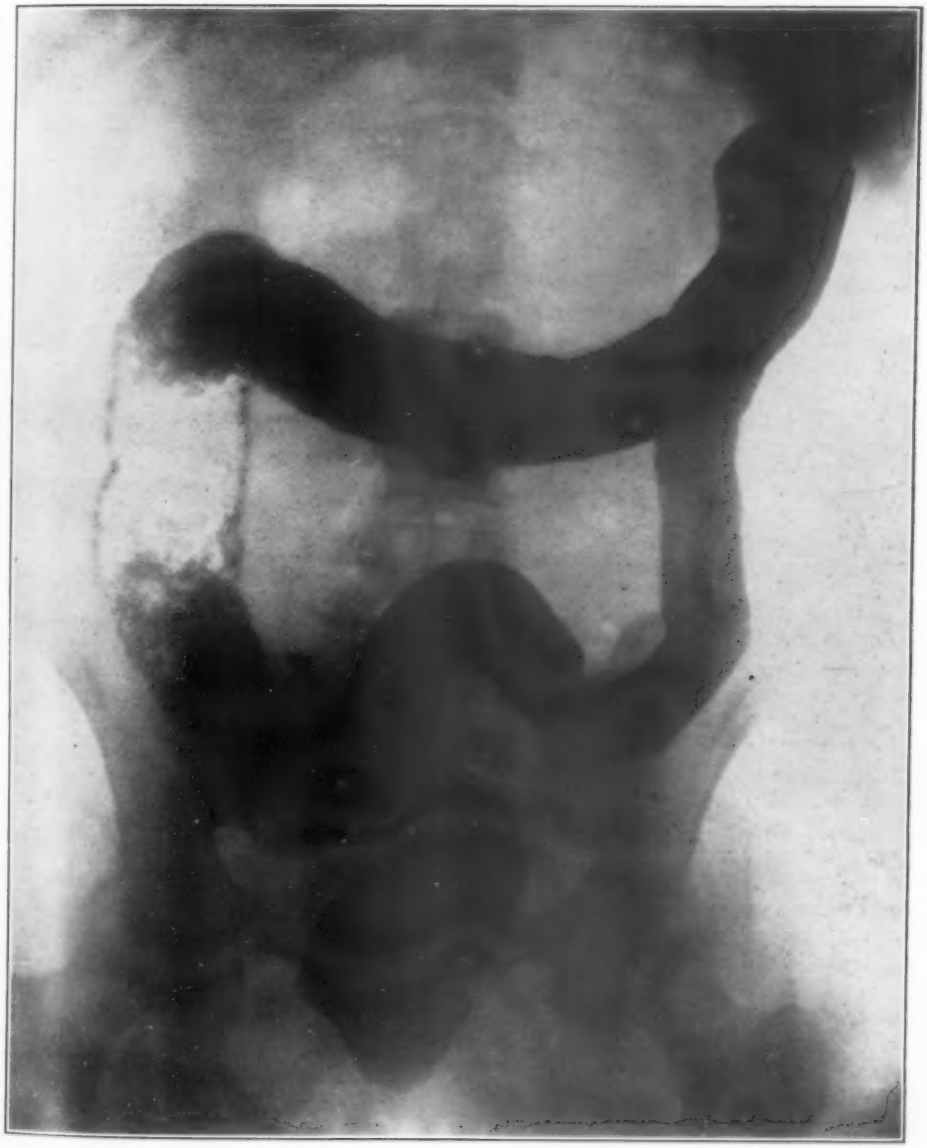

FIG. 22. Case X. Shows increase in the caliber and length of the cecum. Patient now clinically well.

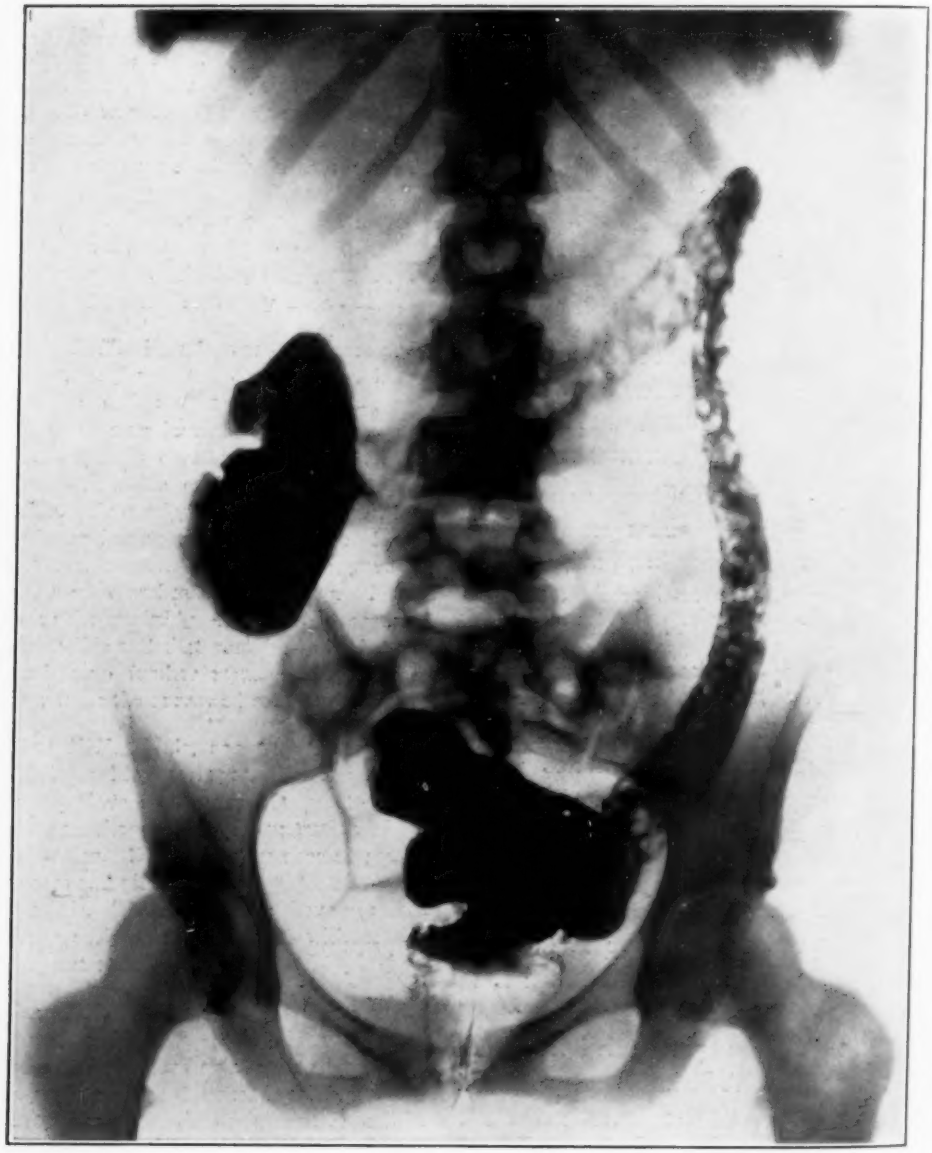

FIG. 23. Case XI. This plate shows remarkable view of the polypoid condition (Dr. Newman secured this plate by a special technic in which the colon was not distended by the barium enema).

FIG. 24. Case XII. Polyposis of the colon. Note irregularities particularly in the transverse colon.

the mucosal folds. Diagnosis: Chronic Colitis. Polyposis of the colon.

This patient had a difficult post-operative course. She developed a fecal fistula. She was capricious with her diet and otherwise obstructive and wholly unco-operative to medical care. Her condition became progressively worse and death occurred at about five months post-operative. The diagnosis of the colonic pathology in this case depended entirely upon the roentgenolocial findings. This was supported and the clinical status of the condition determined by the persistent occult blood reaction of the stools and by the secondary anemia.

Case XIII. Female, age 42. Came under observation June 25, 1923. Case of healed ulcerative colitis discovered accidently in course of examination. Patient came in suffering from constipation and functional disturbances. No evidence of any organic trouble. Proctosigmoidoscopy revealed a membrane having the appearance of healed ulcerative colitis. However, she gave no history of diarrhea or dysentery. I take this to be probably a case that occurred early in life and that completely healed and gave her no further trouble except constipation from the narrowed colon.

Case XIV. Male, age 62. Came under observation May 11, 1925. Duodenal ulcer patient. Gave no history of dysentery. X-ray enema showed loss of haustration of the pelvic colon. Sigmoidoscopy showed granular areas and pitting characteristic of ulcerative colitis. The colitis might have occurred in very early life and healed without any subsequent attacks.

Case XV. Male, age 54. Came under observation Nov. 3, 1911. Had repeated attacks of hemorrhagic proctosigmoiditis. X-ray plate showed normal colon, no deformity characteristic of ulcerative colitis.

Case XVI. Male, age 61. Came under observation May 23, 1921. Lues of the colon.

Case XVII. Male, age 34. Came under observation Nov. 6, 1926. Amoebic dysentery of six months' standing. Note that there is no loss of haustration and that the colon shows contractures here and there.

Conclusion

Bargen's diplococcus is the primary etiologic agent in the production of chronic ulcerative colitis. It must be differentiated from chronic catarrhal form which appears to be a separate disease entity.

The pathology needs revision and further study. The thickening of the wall and contraction of the lumen of the gut does not appear to be due to a true fibrosis. One of our cases revealed a tremendous infiltration of fat in the submucosa.

The autogenous vaccine therapy did not give good results in our series of cases. The best results were secured by hospitalization, blood transfusions, mercurochrome by mouth and rectal injections, and intensive local treatment of lesions in the rectum and lower colon during the convalescent stage. Relapses are frequent and spontaneous cure unquestionably occurs.

Ileostomy should be postponed until all methods of treatment have failed. It is a life saving measure when the lower colon contracts to such a degree that sufficient passage of fecal matter is no longer possible. After ileostomy the colon continues to contract and may be finally obliterated. The cecum dilates and perforation and peritonitis will result if the drainage through the ileostomy opening is insufficient. In cases of this class colectomy is advisable.

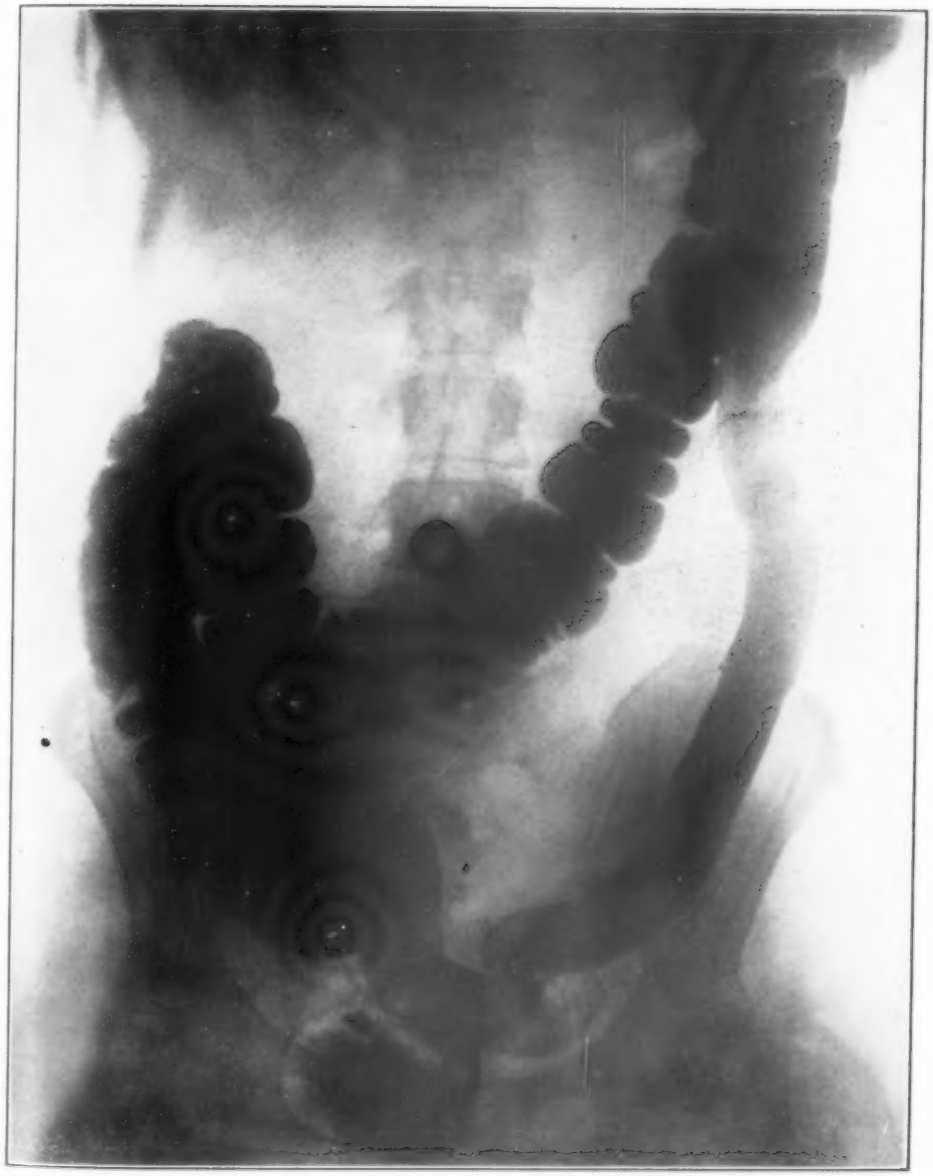

FIG. 25. Case XIII. Case of healed ulcerative colitis. Accidental discovery.

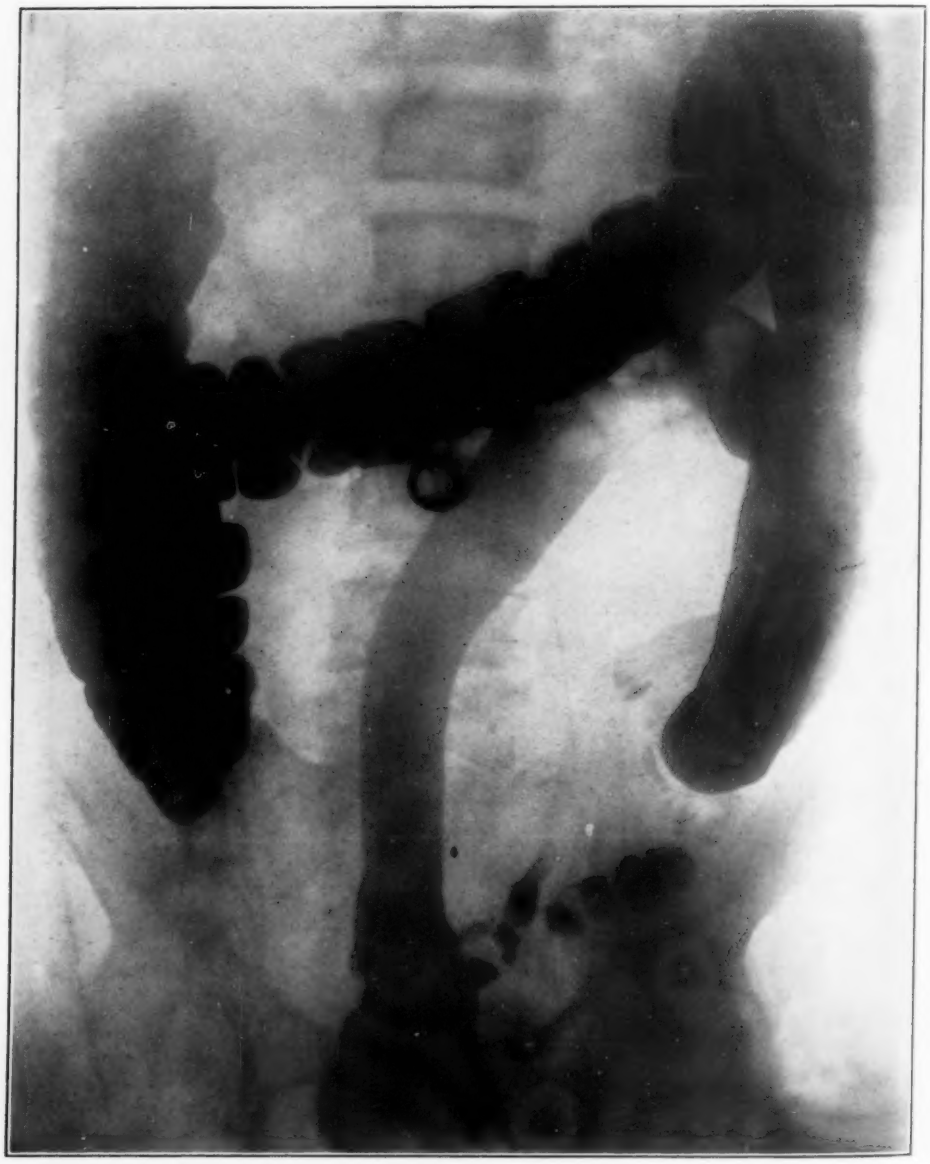

FIG. 26. Case XIV. Case of healed ulcerative colitis. Accidental findings.

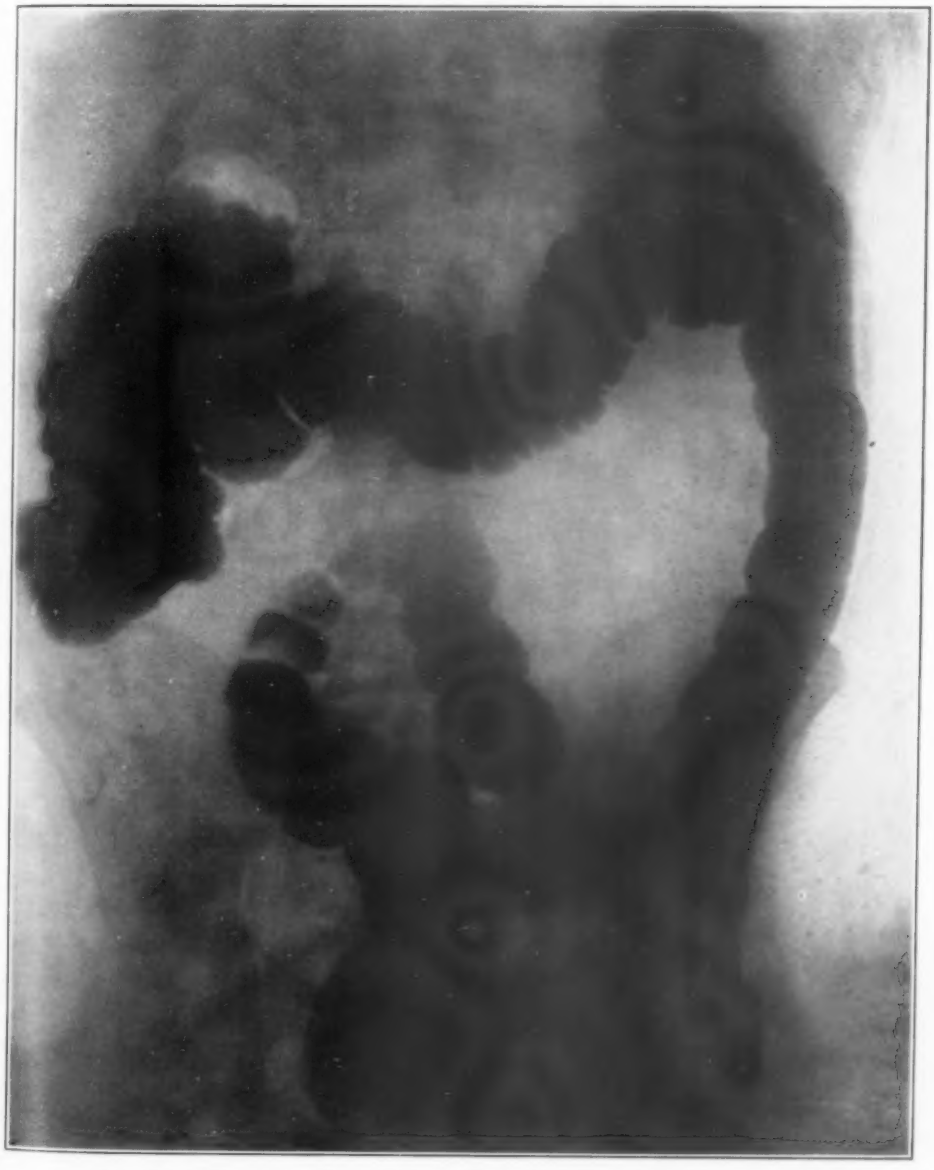

FIG. 27. Case XV. Luetic colitis involving iliac and pelvic colons.

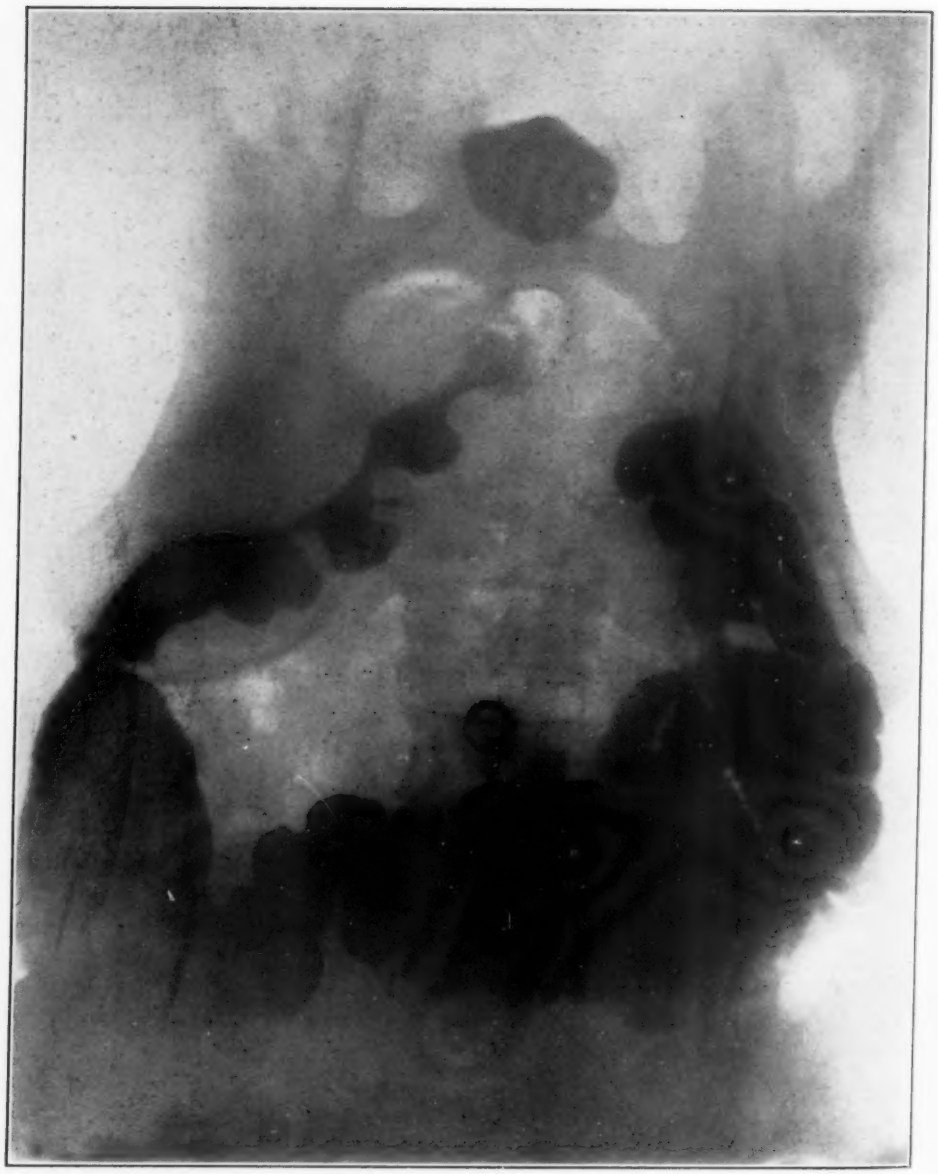

FIG. 28. Case XVI. Case of repeated attacks of hemorrhagic proctosigmoiditis. Normal colon.

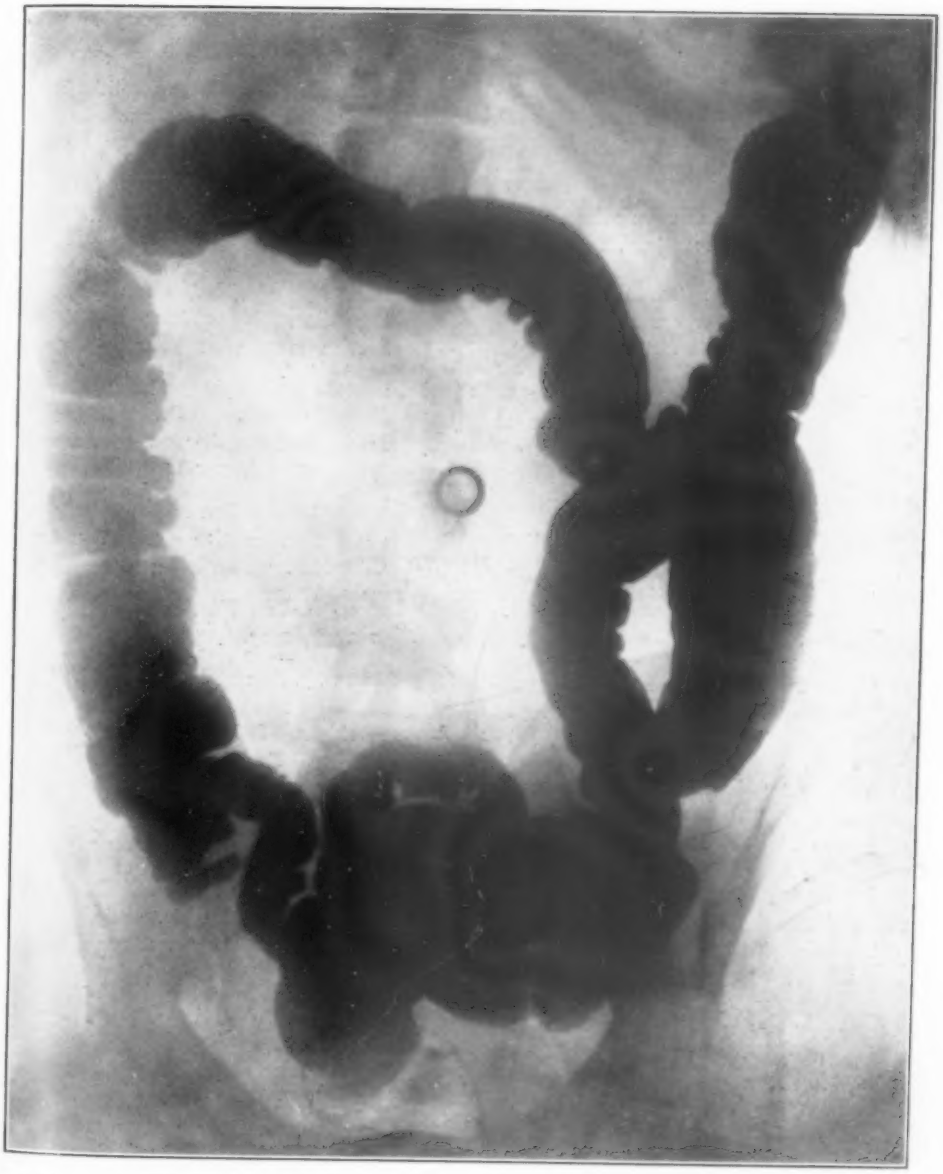

FIG. 29. Case XVII. Amoebic dysentery six months duration. No loss of haustration.

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Editorials

United States Public Health Service

The Surgeon General Reviews World Health Conditions and Summarizes His Work in Annual Report.

AN ACCOUNTING of the health of the people of the United States in so far as Federal duties and responsibilities are concerned is given in the Annual Report of the Surgeon General of the Public Health Service, which has just been transmitted to Congress. This is the Report for the 129th year of the existence of the Service.

The Surgeon General says that through modern developments in public health organization—national and international—it is now practicable for the country to keep informed of the prevalence of epidemic diseases and of the health of populations throughout the world. This knowledge is of considerable importance in preventing the introduction of dangerous communicable diseases into the country from abroad.

Health conditions generally throughout the world were better during the year ended June 30, 1927, than for any previous year on record. The first half of the year was characterized by unusually low death rates in foreign countries. The most serious condition that interposed to prevent even lower mortality rates was the epidemic of influenza which affected a considerable area of that part of the world.

Bubonic plague, which, by reason of its wide geographic distribution and the method of its spread, remains one one of the major quarantinable diseases, continued to be pandemic, but was less prevalent than it has been in recent years. India remained the principal focus of infection, that country having over nine-tenths of the cases reported throughout the world.

Asiatic cholera, which is still a great scourge in India, showed no decrease there, and appeared with more than usual prevalence in the countries eastward as far as Korea and Manchuria.

It is an interesting commentary on the advancement of modern sanitary science that yellow fever—a disease the very name of which not so long ago terrified even the people of this country—was practically confined to one section of the continent of Africa during the year under report. With the exception of one case which occurred at Bahia, Brazil, in July, 1926, the disease was not reported in the Western Hemisphere.

The devastating epidemics of typhus fever which swept Russia after the World War have definitely passed, although the disease remains endemic in that region. There was no marked spread of the disease during the year.

The health of the people of the United States was generally good during the year ended June 30, 1927, as

compared with preceding years. The death rate for all causes for the calendar year 1926 in 28 States was 12.1 per 1,000 population. This was slightly higher than the rate for 1925, which was 11.7. The increase was probably caused principally by the large number of deaths from respiratory diseases. This country escaped the epidemic of influenza which swept over Europe during the winter of 1926-27, and the few cases reported here were mild. Typhoid fever declined during the calendar year 1926, and the case and death rates for diphtheria for the year were the lowest ever recorded. This disease showed a small increase, however, during the first part of 1927. The general downward trend in diphtheria is undoubtedly the result of the use of antitoxin and toxin-antitoxin immunization.

The death rate from tuberculosis continued its decline, and heart disease, diabetes, and nephritis, which had been increasing in recent years, showed lower rates than were expected.

Smallpox decreased during the calendar year 1926. In most sections of this country the disease was of mild type, but in some localities it existed in severe form. Out of a total of 30,450 cases of smallpox reported during the year from 41 States, there were 380 deaths. The disease was the most fatal on the Pacific Coast, which section reported only one-fifth of the total number of cases, but two-thirds of the number of deaths. In view of the demonstrated protective value of vaccination, the Surgeon General says that it is difficult to explain why this simple preventive measure is not universally employed.

Although endemic foci of the important quarantinable diseases continued to exist abroad, no cases of cholera, yellow fever, or bubonic plague gained entrance to the country during the year. Two cases of plague were apprehended aboardship at the United States quarantine station at New Orleans, La., and 17 cases of smallpox and 2 cases of leprosy were detected at domestic quarantine stations. The discovery of three cases of plague among rats at Los Angeles, Calif., however, indicates that the disease continues to exist among rodents; and it persists especially among ground squirrels in certain sections of California, and requires vigilance on the part of the health authorities concerned for the protection of the human population.

The geographical prevalence of tularaemia, a disease identified a few years ago by an investigator of the Public Health Service, was discovered during the year to extend to 10 additional States, increasing the area of known distribution of this new disease to 36 States, the District of Columbia, and Japan.

Rocky Mountain spotted fever is another disease being studied by the Public Health Service, the knowledge of the area of prevalence of which is being extended. This disease originally reported by only two Western States, was reported last year from nine states.

The infant mortality rate (deaths of infants under one year of age per 1,000 live births), considered an important index of the efficiency of public health work, has been decreasing for many years. The rate for 28 States in 1926 was 72.8, as compared with 71.5 for 1925, both of which rates are ex-

tremely low as compared with rates of a few years ago (about 100 in 1915).

The work of the Public Health Service in preventing the introduction of diseases from abroad involves both control at domestic ports and medical inspections at certain foreign ports. At domestic ports during the year, 20,284 vessels, 820,793 passengers, and 1,140,922 seamen were inspected by quarantine officers; at insular ports, 2,991 vessels, 169,461 passengers, and 226,373 seamen were inspected; and at foreign ports, 5,954 vessels, 424,172 passengers, and 272,872 seamen were inspected prior to embarking for the United States. A total of 7,116 vessels were fumigated, either because of the occurrence of diseases aboard or for the destruction of rodents. Thirty-one thousand and seventy-three rats were recovered, of which number 18,334 were examined for plague infection.

The medical examination, by Public Health Service medical officers, of applicants for immigration visas in foreign countries of origin, first inaugurated in 1925, in England, Scotland, and Ireland, was extended during the past year to Germany, Sweden, Norway, Poland and Denmark. The advantages of the system to the prospective immigrants, to the communities of origin, and to the transportation companies resulted in additional requests to the State Department for the extension of the plan to other countries. During the year medical examination of applicants for immigration visas were being made by Public Health Service officers at 20 American consulates in eleven countries. Out of 148,539 applicants examined during the year, 12,974, or 8.75 per cent, were found to have mental or physical disabilities; 6,580, or 4.43 per cent, were refused visas for medical reasons.

At domestic ports 881,699 alien passengers and 996,317 alien seamen were examined under the immigration law, by medical officers of the Public Health Service, and of this number 24,292 passengers and 3,117 seamen were certified for various diseases and disabilities coming under the law.

Investigation was made of the typhoid fever epidemic in Montreal, Canada, with the permission of the Canadian authorities, and a report was made regarding the cause of the epidemic and recommending precautionary measures.

Aid was given the local authorities in the localities of Florida devastated by the hurricane of September, 1926, and the Public Health Service cooperated with the States and the American Red Cross in the emergency relief work in the Mississippi flood area. Within ten days from the beginning of the flood emergency, 14 Public Health Service officers trained in emergency health work and familiar with the localities affected by the flood, were in the field to assist the State Health authorities. Arrangement has also been for a continuance of this cooperation in providing public health protection to the inhabitants of the devastated areas during the period of rehabilitation.

The Public Health Service continued to provide medical and hospital treatment of sick and injured American merchant seamen and other Government beneficiaries—the oldest function of the Service and the specific purpose for which it was organied under the

act of July 16, 1798, 129 years ago—and probably at no time during this period has the aid thus given by the Government to American merchant vessels been of greater value or of more importance in keeping the American flag on the seas.

More than 300,000 beneficiaries apply annually at 152 ports of the United States and its insular possessions where marine hospitals and relief stations are maintained for the giving of hospital and out-patient treatment and making physical examinations. There were 1,288,061 hospital days' treatment given, mostly to merchant seamen and Coast Guardsmen, and 632,341 separate out-patient treatments were supplied. The American seamen are the principal beneficiaries, receiving 67.4 per cent of all the hospital treatment furnished. The Coast Guard furnished 14 per cent of the clientele, while the Employees' Compensation Commission constitutes about one-fifth of the total of the Service beneficiaries.

Because of the exceptional facilities offered in the hospitals of the Public Health Service for the study of syphilis and gonorrhea, important investigations were undertaken, at the marine hospital at New York, of more effective methods of prevention and treatment. Over a period of many years about 20 per cent of all patients treated by the Service were ill as the result of venereal diseases. In 1926, however, this percentage was slightly lower, the rate being 17.

As evidence of the extent of antivenereal disease work during the nine years since the Division of Venereal Diseases was established, 3,105,952 cases of venereal diseases have been reported by the State boards of health, nearly 800 venereal disease clinics have been established by various agencies at which 1,023,326 patients have received 16,330,994 treatments, 62,595 lectures have been delivered to a total of 8,530,000 persons, and 34,676,512 pieces of educational literature have been distributed.

The investigations of public health problems continued during the year. The investigations of stream pollution have been of great practical benefit in determining the limitations of natural and artificial purification of polluted water. The malaria studies have demonstrated the possibility of airplane control of mosquitoes in otherwise inaccessible areas by spreading larvicidal dust. Various inquiries were made in the study of health problems in industry. Other public health problems investigated include child health, posture studies, the advantage of exercise to health, and industrial morbidity.

In investigations of the diseases of man, an extremely important function of the Public Health Service, studies have continued on pellagra, the timely results of which have had a valuable application in the flood area, and on tularaemia, tuberculosis, Malta fever, typhus fever, trachoma, pneumonia, encephalitis lethargica, Rocky Mountain spotted fever, syphilis, goiter, influenza, and leprosy.

Further studies on narcotic drug addiction indicate a reduction in the number of habitues, and confirm the opinion previously arrived at that addiction is, in general, a symptom of neuropsychopathic make-up.

In a survey of salt-marsh mosquitoes conducted in cooperation with the

Bureau of Entomology, it was found that although there are nearly 6,000,000 acres of salt-marsh along the Atlantic and Gulf Coasts, the acreage requiring mosquito control can be expressed in tens of thousands.

It has been the long-standing policy of the Public Health Service to cooperate with official and unofficial bodies for the advancement of the public health. Some of the work is specifically required by law; the remainder is sanctioned by efficient health administration. The volume of cooperative work conducted during the fiscal year was extensive, and included practically all the departments and some independent establishments of the Government.

In the field of international developments, the year was marked by two important international official conferences for the improvement of existing treaties or conventions regulating maritime procedures. The representatives of 60 Governments, including the United States, met during the year at Paris to revise the International Sanitary Convention of Paris (1903, 1912), which was necessitated by recent advances in medical science. A conference of the directors of health of Pan American Governments was held in Washington, under the chairmanship of the Surgeon General as director of the Pan American Sanitary Bureau, during which constructive progress was made in matters pertaining to Pan American public health and sanitation.

Among the recommendations made by the Surgeon General in his annual report, he commends a bill introduced in the last Congress for the coordination of public health activities of the Government, which provides for a grant of authority to the President to transfer to the Public Health Service any executive agency (other than those in the War and Navy Departments and those in the Veterans Bureau) when such transfer is deemed to promote greater efficiency in the conduct of public health work. The bill also provides for details of officers of the Public Health Service to other executive agencies, upon request of the heads of departments or independent establishments; for the extension of research through close cooperation with educational and research institutions by an enlarged hygienic laboratory; and for the coordination of research of public-health officials and scientific workers. Provisions were also contained for unification of terms of appointment of personnel, with compensation and tenure assured to scientific and professional workers adequate to attract to the service, men and women of proved qualifications; and for the enlargement of the present advisory board for the hygienic laboratory into a national advisory health council. The Surgeon General believes that such a coordination of public health activities would tend to eliminate duplication of effort in administration, research, and educational measures, and would prove a decisively forward step in public health in the United States.

Abstracts

Hypertension of the Pulmonary Circulation. Its Causes, Dynamics and Relation to Other Circulatory States. By ELI MOSCHOWITZ (Amer. Jour. of Med. Sc., Sept. 1927, p. 388).

Hypertension of the lesser circulation is common. It passes generally under the name of "pulmonary congestion," "right-sided insufficiency," or arteriosclerosis of the pulmonary vessels." It is caused by any lesion that increases the peripheral resistance within the lesser circulation. The most common causes are mitral disease, especially mitral stenosis, emphysema, whether primary (senile) or secondary (asthmatic), infiltrating lesions of the lung (chronic tuberculosis with induration, bilateral pleural synechiae, chronic interstitial pneumonia, tumors), kyphoscoliosis, patent ductus arteriosus and communications between the two sides of the heart. A sustained hypertension of the lesser circulation leads to arteriosclerosis of the pulmonary vessels. A "primary" sclerosis of the pulmonary vessels, if it exists at all, is extremely rare. There is an interdependence in the incidence of arteriosclerosis of the greater and lesser circulations. The compensatory dynamics following hypertension in the lesser circulation are described. The physical signs of hypertension in the lesser circulation are those brought about by the compensatory mechanism. In the main these are: Increased venous pressure, accentuation of the second pulmonic sound, dilatation and hypertrophy of the right heart, dilatation of the pulmonary conus, cyanosis, dilatation of the superficial veins (especially pectoral), enlargement and tenderness of liver, lowered kidney function and infarction of the lungs. It is the hypertension and not the arteriosclerosis that is responsible for this clinical syndrome. As in the greater circulation, it is the disturbed function and not the anatomic sequence that is at fault. A transient (usually) terminal hypertension of the lessor circulation arises under a number of other conditions. In general vascular hypertension, hypertension of the lesser circulation may follow mitralization, when myocardial insufficiency sets in and when there is an associated emphysema. This explains, in part, the difference between the pale and the cyanotic hypertensive. In left coronary artery disease hypertension of the lesser circulation arises only after myocardial insufficiency sets in. The so-called "Ayerza's disease" is not a disease, but a syndrome developing from any lesion that causes hypertension and consequent arteriosclerosis of the lesser circulation. The constant relation of syphilis to this disease is not proven. Edema of the lungs may either follow or cause hypertension of the lesser circulation. Changes in cardiac rhythm cause hypertension of the lesser circulation only when myocardial insufficiency arises. There is a likelihood that cirrhosis of the liver associated with cardiac disease is the result of the increased venous tension within the hepatic area and of an hepatic arteriocapillary fibrosis. Hypertension of the lesser circulation may be the mechanism of the hitherto unsatisfactorily explained cyanosis in congential heart disease.

An Experimental Study of Diathermy, IV. Evidence for the Penetration of High Frequency Currents Through the Living Body. By RONALD V. CHRISTIE AND CARL A. L. BINGER (Four. of Exper. Med., November 1, 1927.)

In this fourth paper on an experimental study of diathermy the principles governing the passage of high frequency currents through various conductors are discussed and exemplified in the experiments done on both non-living and living bodies. It is

shown that the current takes the path of least electrical resistance rather than the shortest path, and the maximal heating occurs at the point of greatest concentration of the lines of the current flow. In a homogeneous medium with parallel electrodes maximal heat production occurs in those portions of the medium adjoining the electrodes and the heat gradient is from without inward. Under these circumstances maximal heating never occurs at the center. In discussing the localization not only the electrical resistance and current concentration, but also the cooling effect must be considered. In experiments on the dog's cavader no evidence of the so-called "skin effect" could be demonstrated. This is in coutradistinction to the findings of Bertman and Crohn, but the discrepancy is explained on the ground of a technical error in their work. The finding of no "skin effect" is in harmony with the conclusions of Donne and Tredell, based on both experimental and theoretical considerations. As the result of experiments on the anesthetized dog, the conclusions drawn were: 1st. The heat gradient of the body is reversed during diathermy and heating occurs from without inward. 2nd. Deep heating during diathermy is greater than that which results from the application of local heat to the skin. 3rd. The lung can be heated by diathermy in spite of simultaneous cooling of the chest wall. The authors regard their experiments as giving satisfactory evidence of the passing of the current through the interior of the body.

Degenerative Changes in the Germinal Epithelium in Acute Alcoholism and their Possible Relationship to Blastophthoria. By CARL VERNON WELLER (Trans. of the Assoc. of Amer. Physicians, Vol. xlii, p. 277, 1927).

Weller has studied the pathological changes occurring in the testes of nine cases of acute alcoholic intoxication, all of them being coroner's cases in which death occurred during, or immediately after severe alcoholic intoxication. One of these, in a boy of 17 years, was a first experience; the others were probably an exacerbation of a more or less chronic or intermittent alcoholism. In all of these cases the germinal epithelium showed marked parenchymatous degeneration, leading when sufficiently marked to total aspermatogenesis, the various orders of maturing cells disappearing in an inverse order to that of their production and the basal layer of cells alone remaining to afford the possibility of regeneration if the individual survives. In the testes showing less marked degeneration there is an apparent increase in spermatocytes and spermatids, with very few tailed spermatozoa, as if there had been an inhibition in the final steps of spermatogenesis. With somewhat more marked changes the germinal epithelium becomes thinner than normal, perhaps only two to four cells thick. At this stage there are but few spermatids, and the spermatogonia and spermatocytes take on a marked degree of vacuolation, which may have a zonal distribution in the tubal epithelium, the spermatids next to the lumen escaping. A typical cell division forms, teratocytes are numerous, appearing as mutinucleate masses near the lumen, apparently spermatids which have accomplished nuclear division only, and giant nuclear forms which are found especially in the deepest layer of the germinal epithelium next to the basement membrane. Finally the epithelium becomes reduced to one or two rows only, and no evidence of spermatogenesis is present. That portion of the germ plasm which is in process of maturation is especially susceptible. The survival of the, as yet, undifferentiated primary germinal epithelium makes possible the restoration of spermatogenic function following nonlethal intoxication. The changes found are in no sense specific for alcoholic intoxication, precisely similar ones are found clinically in a great variety of conditions, particularly in epidemic pneumonia, and they can be duplicated in animals in experimental lead poisoning. There is no proof that alcohol acts directly upon the testis; while this seems probable the primary changes may be elsewhere. Such blastotoxic conditions, particularly the alcoholic because of its frequency, become of great bio-

logic and clinical importance. The testicular changes found are in excess of those which it is necessary to produce experimentally in order to demonstrate a deleterious blastophthoric effect by breeding experiments. It is certain that in the earlier stages spermatozoa capable of giving rise to defective off-spring must be set free. It follows that procreation during a period of intoxication entails a definite hazard as to the quality of the off-spring which may result. It has been shown that this applies to lead poisoning as well as to alcoholism, pneumonia, typhoid fever, certain dietary deficiency diseases, exposure to certain forms of radiant energy, etc., a list which is being constantly increased. Thus this conception of the liability of the germ plasm is capable of extensive practical eugenic application.

Antirachitic Value of the Sun's Rays Through Various Special Window Glasses. By Frederick F. Tinsdale and Alan Brown (Amer. Jour. of Diseases of Children, November, 1927, Vol. xxxiv, p. 742).

This research was carried out under a grant from the Department of Health of the Province of Ontario. Three glasses: 1. Vitaglass, manufactured in England, 2. Vioray glass, 3. Corning Special glass, which have been claimed will transmit the antirachitic rays of sunshine were experimentally tested out with animals placed on a rachitogenic diet (McCollum's). As a result of their experiments these investigators concluded that the antirachitic effect of the sun's rays (including skyshine) through Vitaglass, Vioray glass and Corning special glass is from 25 to 50 per cent of the antirachitic effect obtained without the use of glass. Obvious differences could not be detected in the efficiency of the three glasses. No antirachitic effect, or at most a negligible one, is produced by the sun's rays through ordinary glass. The antirachitic effect of skyshine through an ordinary window glazed with Vioray glass is slight—in fact almost negligible except immediately adjacent to the window. Similar results were obtained with rays through an open window covered with ordinary fly screen. In order to obtain much benefit from rays through an open window, or a window glazed with special glass, it is necessary to receive the direct rays of the sun. The use of special glass during the winter months is probable of little value. Its use is justified in the latitude of Toronto from about the first of March on, as the inclement spring weather prohibits the exposure of patient to the sun's rays which at that time have a great antirachitic effect.

The Incidence of Scarlet Fever Streptococci in Throats of Diphtheria Patients. By Paul S. Rhoads (Jour. of Infectious Diseases, November 5, 1927, p. 377).

In spite of the most rigid precautions against cross infections scarlet fever sometimes develops in patients admitted to diphtheria wards. Councilman, Mallory and Pearce in a study of 220 deaths from diphtheria found scarlet fever 34 times. It has long been known that streptococci could be cultured from the complicating lesions of diphtheria more often than any other micro-organism. Until methods of recognizing the scarlatinal streptococci have been worked out, it was impossible to know how many of the complications were due to scarlatinal infection. Interest in the problem was stimulated by the observation that occasionally a diphtheria patient who reacted positively to the Dick test on admission, became negative before leaving the hospital without developing a recognized rash. At times such patients have desquamated. The question naturally arises whether or not these may have been diphtheria patients or diphtheria carriers who were at the same time suffering from throat infections due to scarlet fever streptococci, but without a rash, or having a rash so transient that it was not observed. That scarlatinal throat infection may occur without eruption has long been suspected by clinicians. Dick and Dick showed experimentally in 1921 that such infections do occur. In 1925, Williams using the toxin neutralization method described by Dick and Dick identified 42.8 per cent of hemolytic streptococci from excised tonsils as well as one strain from the sputum of a

patient with bronchitis and one from an osteomyelitis lesion as scarlatinal strains. Stevens and Dochez using the same method found 5 scarlatinal strains in the 17 hemolytic streptococci tested. These strains came from the throats of nurses with acute pharyngitis during an outbreak of scarlet fever in the nursing group. With agglutination and agglutinin absorption methods their percentage of scarlatinal strains was slightly higher. Nicholls studied 21 strains from sources other than scarlet fever patients, most of them from patients with tonsillitis, pharyngitis or sinusitis. Using the toxin neutralization method ten of these were identified as scarlatinal strains. Rhoads' study was that of the incidence of hemolytic streptococci in the throats or noses of 100 patients admitted with a diagnosis of diphtheria to Chicago hospitals for contagious disease, and then to determine which of the hemolytic strains were scarlatinal or non-scarlatinal by the toxin neutralization method. As a result of this study hemolytic streptococci were cultured from the throats or noses of 29 of a series of 100 patients who were admitted with the diagnosis of diphtheria to hospitals for contagious disease in Chicago. Sixteen or 55.2 percent of these strains of hemolytic streptococci were identified as scarlet fever streptococci by the toxin neutralization method. The strength of the scarlatinal toxin produced by these strains varied from 100 to 10,000 skin test doses per cc. It was less than 2,000 skin test doses per cc. in 14 of the 16 strains. Thirteen, or 44.8 per cent, of the strains of hemolytic streptococci isolated did not produce any demonstrable scarlet fever toxin. It is possible, therefore, that infection with scarlet fever streptococci of comparatively low toxin production may account for the development of a negative Dick test during the course of acute infections diagnosed clinically or culturally as diphtheria. It is possible that some of the cases reported were scarlet fever occurring in diphtheria carriers and diagnosed diphtheria because of positive cultures. The results reported indicate the advisability of individual isolation to present cross infection with hemolytic streptococci in diphtheria wards.

Reviews

The Methods of Clinical Diagnosis. By ALEXANDER GEORGE GIBSON, M.D., F.R.C.P., AND WILLIAM TREGONWELL COLLIER, M.D., M.R.C.P., 398 pages, 4 plates. Longmans, Green & Co., New York, 1927. Price in cloth, $5.00.

The object of this book is to supply the student with a short practical guide to the fundamentals of disease. The aim of the book throughout is to give a knowlege of the technique of examination, what to look for and how to look for it. The student is asked to recognize the importance of a routine examination of the patient from top to toe. More errors are due to the omission of some part of the examination than to misinterpretation of the signs discovered, and although in the stress of practice some part of an examination may have to be discarded, it can never be discarded safely. From this point of view the book has been divided into two sections, the examination of regions and the examination of systems, so that a general review of the whole body should be carried out before attention is concentrated on the system believed to be primarily affected. The authors have also endeavored to emphasize the importance of the simpler methods of examination. Percussion and auscultation should always follow not precede inspection; instrumental and laboratory methods should always come last. For this reason they have added to each system an outline of symptoms, since in many cases more is to be learned from the history than from physical examination. An attempt is made to teach the student the habit of making inferences from what he sees, and therefore of making provisional diagnoses as he goes along, which may be dealt with when all the evidence of the examination is available. If the student studies the methods by which the most skilled of his teachers arrive at a diagnosis he will be struck by the stress laid on certain parts of the examination and the slight attention paid to others. This art of knowing what to omit is the result of prolonged experience, and a conscious or unconscious judgment in what is important and what is trivial. This may appear to be an intuitive gift, but it cannot be developed apart from practice. The student is advised to read first the examination of Regions; the chapters dealing with Systems should be read in conjunction with actual cases. The consideration of Chapters I and II may be left until the technique has been mastered. In dealing with clinical pathology the aim has been to give one reliable method rather than a choice, and to restrict all methods to those that can be done by the student or practitioner himself without access to a well-equipped laboratory. There are seventeen chapters, the first four dealing with an elementary introduction concerning aims of diagnosis and nature of disease, the general methods of diagnosis, general features of the patient and the examination of regions; the remaining chapters deal with the various systems. This is a very good manual for the student who is taking his course in physical diagnosis; it will also be a great aid to him during his interneship, and will aid the general practitioner by refreshing his mind in regard to matters of technique and conditions that should be looked for.

The Normal Diet. A Simple Statement of the Fundamental Principles of Diet for the Mutual Use of Physicians and Patients. By W. D. SANSUN, M.S., M.D., F.A.C.P., Director of the Potter Metabolic Clinic, Department of Metabolism, Santa Barbara Cottage Hospital, Santa Barbara, California. Second edition. 136 pages.

C. V. Mosby Company, St. Louis, Missouri. Price in cloth, $1.50.

The author has for many years given the subject matter of this book in lecture form to patients suffering from the various nutritional disorders. Since diet errors are very common, and such errors are undoubtedly responsible for many minor ailments, as well as for some of the more serious ones, he believes that a simple statement of the fundamental principles underlying the selection of a normal diet may fill a definite need. Normal diet menus are given as well as special menus to illustrate how such diets may be built without violating any of the fundamental principles. Chapter II treats of the bulk requirements of the body, Chapter III of the acid-ash type of acidosis, Chapter IV of the acetone type of acidosis, Chapter V of the caloric requirements of the body, VI of the protein requirements, VII of the mineral, VIII of the vitamine and IX of the water requirements. This little volume is devoid of fads, and the material given is consistent with our scientific knowledge of the subject. It is simply and clearly written, and is a book of value for the household as well as for the hospital diet kitchen.

The Diabetic Life. Its Control by Diet and Insulin. A Concise Practical Manual for Practitioners and Patients. By R. D. LAWRENCE, M.A., M.D., M.R.C.P., (London); Chemical Pathologist and Lecturer in Chemical Pathology, King's College Hospital. Third edition. 185 pages, 11 illustrations. P. Blakiston's Son and Company, Philadelphia, 1927. Price in cloth, $2.50.

A third edition of this work has been called for within a year of the appearance of the second. During this time there have been no revolutionary ideas promulgated concerning insulin or the treatment of diabetes. New practical details have, however, been incorporated in this edition as the result of increased experience, and a full explanation of the mode of insulin action and the ketone ratio have been added. The object of the book is to bring the modern treatment of diabetes by diet and insulin within the scope of the general practitioner and the understanding of the patient, whose intelligent cooperation is necessary for the best results. Sufficient knowledge has accumlated to make it possible to lay down simple rules of treatment without being too dogmatic. By means of a simple yet varied and accurate diet scheme—the Line-ration diet—the busiest practitioner is enabled to start accurate treatment without any elaborate calculation of diets and food-stuffs. It is a book for both patient and doctor. The discussion of the theoretical basis of treatment has been made as short as possible, and the routine treatment has been dealt with in full practical detail. The book is, of course, written from the standpoint of insulin chiefly. It is, however, sufficiently comprehensive, is well written, and one of the best of the special manuals written upon the subject. It may be highly recommended.

Diseases of the Skin. By HENRY H. HAZEN, A.M., M.D., Professor of Dermatology in the Medical Department of Harvard University; Sometime Assistant in Dermatology in the Johns Hopkins University; Member of the American Dermatology Association. Third edition. 572 pages, 248 illustrations including two color plates. C. V. Mosby Company, St. Louis, 1927. Price in cloth, $10.00.

The first edition of this book appeared in 1915, the second in 1922. This, the third edition, contains a number of radical changes. Diseases have been classified more accurately according to their etiology. In accordance with modern beliefs the word "Eczema" has been omitted. The subject of Anaphylactic Dermatitis has received considerable mention. The use of X-rays, radium, unipolar and bipolar fulguration and the Alpine lamp has been carefully revised. The bibliography has been omitted, but a number of new illustrations have been included; the great majority of the photographs were taken either in the author's own clinic or in that of Dr. Gilchrist. A

very large proportion of these illustrate skin diseases in the negro. The paucity of photomicrographic illustrations is notable. Many of the gross photographs are not very well produced, and from the standpoint of illustration this work is much inferior to other recent publications on Dermatologic affections. The text-material is also much reduced; some subjects are insufficiently treated. Syphilis is discussed from the dermatologic standpoint. The most individual thing about this book is that it offers an unusual amount of material concerning dermatologic conditions in the negro.

College News Notes

THE JAMES M. ANDERS TESTIMONIAL DINNER

Celebrating His Fiftieth Anniversary in the Practice of Medicine

Tuesday, November 29, 1927, at 7 o'Clock

A testimonial dinner was given Dr. James M. Anders of Philadelphia on November 29 in honor of his fiftieth anniversary in the practice of medicine. Dr. Josiah H. Penniman, President and Provost of the University of Pennsylvania, presided at the dinner, which was attended by a large number of prominent members of the medical and legal professions. Dr. John B. Deaver, on behalf of those present, presented a life-size bust of Dr. Anders to the doctor.

Dr. Anders has the distinction of being the only individual on whom has ever been conferred Mastership in the American College of Physicians. As President of The College during 1922-23, and a member of the Board of Regents for three years, he rendered most valuable service in determining many of the present policies and principles of the organization.

He is an author and has long been active in civic and welfare work. From 1882 to 1916 he was professor of medicine and clinical medicine in the Medico-Chirurgical College of Philadelphia, and thereafter professor of medicine in the Graduate School of Medicine of the University of Pennsylvania. He is now a member of the Board of Managers of the Medico-Chirurgical and Polyclinic Hospitals, and a director of the Health Council and Tuberculosis Committee.

Dr. Anders is an ex-president of the Philadelphia County Medical Society, member of the Pennsylvania State Medical Association, fellow of the American Medical Association, ex-president of the American Climatological and Clinical Association, ex-president of the American Society of Tropical Medicine, ex-president of the American Therapeutic Society and a member of the Alpha Mu Pi Omega medical fraternity.

Dr. William R. Bathurst (Fellow, February 21, 1924), of Little Rock, Ark., is the newly elected President of the Southern Medical Association. Dr. Bathurst is a dermatologist who has distinguished himself not only in his own state but elsewhere. He is professor of dermatology at the University of Arkansas School of Medicine and a member of the staffs of St. Vincent's Infirmary, the General Hospital and the Missouri Pacific Hospital. In addition, he is editor of the Journal of the Arkansas Medical Society.

The Southern Medical Association will hold its Annual Meeting for 1923 at Asheville, N. C.

OBITUARY

Dr. Robert L. Crum died following a stroke of apoplexy on the 26th of October at his home in Los Angeles. Born in 1881, he received his medical education at St. Louis University. He was a member of the Hollywood Country Club, the University Club and of the Los Angeles County, California State and the American Medical Association. Since February 1920 he has been an Associate of the College. He was on the Staff of St. Vincent's and the Los Angeles General Hospitals. He was a Captain in the Medical Corps during the World War. He was a thirty-second degree Mason and a Shriner.

The members of the American College of Physicians and his many professional friends mourn the passing of a good physician.

PRELIMINARY PROGRAMME FOR NEW ORLEANS MEETING

Preliminary program of the American College of Physicians for the meeting in New Orleans next spring, beginning March 5. The meeting will start on Monday, at 10 A. M. with the usual address of welcome from the Mayor of New Orleans; the President of Tulane, Dr. Dinwiddie; The President of the Orleans Parish Medical Society and Dr. C. C. Bass, Dean of the School of Medicine of Tulane University of Louisiana. Dr. Frank Smithies will give a reply to the address of welcome. The program will be continued as follows:

1. Dr. Julius Bauer, Vienna, Austria. (Title to be announced).

2. Dr. David P. Barr, "Multiple Myeloma."

3. Dr. L. G. Rowntree and Dr. George E. Brown, "Studies in Blood Volume with the Dye Method."

4. Dr. Joseph Sailer. Title to be announced.

5. Dr. Frank R. Menne, "The Effect of Iodine on the Histopathology of the Thyroid Gland in the Instance of Hyperthyroidism."

Evening Session, Monday, 7:45

1. Dr. Maud Slye, "Cancer and Heredity."

2. Dr. J. L. Goforth, "Natural and Acquired Body Resistance to Neoplasia."

Tuesday Morning, March 6—10 o'Clock

1. Dr. Charles T. Stone, "The Occurrence of Severe Anemia in Myxedema."

2. Dr. W. W. Duke, "Diagnosis and Treatment of the Anemias."

3. Dr. Hilding Berglund, "Liver Diet in Pernicious Anemia."

4. Dr. C. C. Sturgis, Dr. Raphael Isaacs and Dr. Millard Smith: "Treatment of Pernicious Anemia with Liver Fraction."

Symposium on Tuberculosis.

5. Dr. Charles L. Minor. Title to be announced.

6. Dr. F. M. Pottenger, "The Cause of the Varied Clinical Manifestations in Pulmonary Tuberculosis."

7. Dr. Gerald Webb. Title to be announced.

8. Dr. Robert S. Berghoff, "Intestinal Tuberculosis."

9. Dr. John W. Flinn, "A Study of the Differential Blood Count in One Thousand Cases of Active Pulmonary Tuberculosis."

Evening Session—7:45 o'Clock

1. Dr. James S. McLester. Title to be announced.

2. Dr. Allen K. Krause, "The Pathogenesis of Tuberculosis."

3. Dr. T. Z. Cason, "Some Unfinished Research Problems of the South."

Wednesday Morning, March 7—10 o'Clock

1. Dr. L. F. Bishop, "The Practice of Cardiology."

2. Dr. Morris H. Kahn, "Heart Strain and its Consequences."

3. Dr. J. P. Anderson, "Discussion of the Diagnosis of Coronary Occlusion with Special Reference to Its Simulation of Acute Abdominal or Other Surgical Conditions, with Illustrative Cases."

Symposiun on Epilepsy

4. Dr. H. Rawle Geyelin, "The Relation of Chemical Influences, Including Diet and Endocrine Disturbances, to Epilepsy."

5. Dr. E. Bates Block, "The Relation of Organic Brain Disease to Epilepsy."

6. Dr. T. H. Weisenburg, "The Relation of Extra-cranial Disease to Epilepsy."

7. Dr. Tom Throckmorton. Title to be announced.

Evening Session—7:45 o'Clock

Symposium on Infectious Diseases.

1. Dr. C. E. Birkhaug, "Erysipelas."

2. Dr. A. R. Dochez, "Scarlet Fever."

3. Dr. Jean V. Cooke, Specific Prophylactic Measures in Varicella and Measles."

4. Dr. J. C. Small, "Rheumatic Fever."

THURSDAY MORNING, MARCH 8—10 O'CLOCK

1. Dr. C. Saul Danzer, "The Pathogenesis and Treatment of Dyspnoea in the Light of Recent Experiments."

2. Dr. T. G. Schnabel, "A High Fat Diet in the Treatment of Migraine."

3. Dr. Henry Wald Bettmann, "Chronic Appendicitis from the View Point of an Internist."

Symposium on Diabetes.

4. Dr. Anthony Bassler, "Chronic Pancreatic Disorders, Diabetic and Non-diabetic."

5. Dr. Frederick M. Allen, "Present Results and Outlook of Jealetic Treatment."

6. Dr. A. A. Herold, "Diabetic Therapy, with Special Reference to the Newer Remedies."

7. Dr. W. H. Olmstead. "The Dietetic Management of the Diabetic in the Doctor's Office."

FRIDAY MORNING, MARCH 9—10 O'CLOCK

Symposium on Tropical Medicine.

1. Colonel Roger Brooke. Title to be announced.

2. Dr. Aldo Castellani. Title to be announced.

3. Dr. Wm. M. Jones, "The Early Lesions of Intestinal Amebiasis."

4. Dr. Bailey K. Ashford, San Juan, Porto Rico. Title to be announced.

5. Dr. Aristides Agramonte, Havana, Cuba. Title to be announced.

FRIDAY EVENING, MARCH 9—7:45 O'CLOCK

1. Dr. Julius Bauer, Vienna, Austria.

Convocation Address.

The scientific program as given above will take about half of the time of the meeting. The remainder of the time will be occupied by clinics. In a general way I will give you our plan of providing clinics. In the afternoon from 1:45 to 4:30, clinics will be held at the Charity Hospital of New Orleans, Touro Infirmary, the Hutchinson Memorial, the Richardson Memorial, Hotel Dieu, the Baptist Hospital, Mercy and Presbyterian Hospitals. At the Charity Hospital and Touro Infirmary, the program will be changed in the middle of the week, so that two separate programs will be put on at these two hospitals. The same will apply to the program at the Hutchinson Memorial. Clinics will be given at the Charity Hospital by Dr. George Bel, Dr. J. B. Guthrie, Dr. Amadee Granger, Dr. A. E. Fossier, Dr. J. L. Lewis, Dr. Randolph Lyons, Dr. J. H. Musser, Dr. Robert Bernhard, Dr. W. A. Love, Dr. George R. Herrmann, Dr. Philip Jones, and others. At the Charity Hospital, Dr. Julius Bauer, of Vienna, will give a clinic on certain days.

At the Touro Infirmary, clinics will be given by Dr. L. R. DeBuys and his staff and by Dr. I. I. Lemann and his staff. Dr. DeBuys' staff will limit themselves to diseases of children; Dr. Lemann and his staff to internal medicine and to subjects related to internal medicine more or less closely, so that it is quite possible with the present plan to have a surgeon to discuss cases in which the internist and the surgeon come into close contact, such as thyroid cases, abdominal conditions and so on.

At the Baptist Hospital, Dr. Oscar W. Bethea will run the program.

At Hotel Dieu, Dr. S. Chaille Jamison, Dr. Maurice Couret, Dr. L. A. Fortier and others will give clinics.

At the United States Marine Hospital, Dr. W. C. Rucker will be in charge of the program. There are always a large number of interesting tropical and unusual diseases at the Marine Hospital.

At the Hutchinson Memorial, clinics and demonstrations will be given daily. Dr. Wm. M. James and Dr. J. J. Vallarino, of Panama, will demonstrate their preparations and x-ray findings in amebiasis. Dr. J. C. Small, in addition to his scientific paper, will give in more detail than can be done at a large scientific meeting the particulars of the studies he has made in rheumatic fever. Dr. Robert S. Berghoff will give an afternoon demonstration on the diagnosis of chest diseases; Dr. T. J. Perkins, "Constitutional Type in Relation to Mental Disease;" Dr. Aldo Castellani, "Fungus Growth;" Dr. C.

C. Bass, "Malaria;" Dr. F. M. Johns; Dr. H. W. Butler, "A Slide Method for the Diagnosis of Syphilis;" Dr. Roy H. Turner, "Intestinal Microbiology." A clinical pathological conference will be given one or two days.

The program at the Richardson Memorial will be under the supervision of Dr. J. A. Lanford, who has invited Dr. C. W. Duval, Dr. Henry Laurens, Dr. Irving Hardesty and Dr. J. T. Halsey, Professors of Pathology, Physiology, Anatomy and Pharmacology, respectively, with their staffs, to demonstrate research problems that they are actively engaged in or which they recently completed.

Reduced Fares

Twelfth Annual Clinical Session, New Orleans, La.

The Executive Secretary advises that all railroads of the United States and of eastern Canada have granted reduced fares to our Clinical Session on the Certificate Plan of fare and half fare. Those who attend the Session will purchase going ticket at local railroad office and at the same time request a "Certificate." This Certificate, when validated by the proper officers at the registration booth at New Orleans, will entitle attendant to purchase return ticket a half rate.

These reduced rates apply not only to the attendant, but to dependent members of his family.

Going tickets may be purchased from March 1 to 7, and the return ticket may be used up to March 13. For those who desire to remain in New Orleans longer, the return ticket may be used until March 24, upon deposit of Certificate with W. H. Howard, Special Agent located in the City Ticket Office of the Louisville and Nashville Railroad, New Orleans, upon payment of fee of $1.00 per Certificate receipt at time of deposit.

Special Trains

Twelfth Annual Clinical Session, New Orleans, La.

The Southern Railway System, in conjunction with other railroads of the East, have announced the "Eastern Physicians' Special" train starting from Boston, March 3d, passing through New York, Philadelphia, Baltimore, Washington, to arrive at New Orleans, Monday morning, March 5th. They also announce the "Central States Special," serving eastern Canada and eastern Central States, with connections at Cincinnati and joining the "Eastern Physicians' Special" at Chattanooga and continuing to New Orleans.

Of especial interest is the arrangement for a special train leaving New Orleans after the Convocation on Friday night, March 9th. Heretofore, it often became necessary for many who wished to attend the Convocation to remain over night, because of no suitable train accommodations being available after the Convocation Exercises. This special train has been arranged especially for the convenience of all members who attend the Convocation and wish to leave for the northeastern section of the country immediately thereafter. Time-tables are available in the Executive Secretary's office, and will be distributed to all members residing in the territories served by these special trains.

The route traversed includes the Piedmont Section of Virginia, Blue Ridge Mountain Range, the valleys of the Holston and the Tennessee Rivers, Historical Chattanooga, Industrial Birmingham and across Lake Pontchartrain.

Members residing in the territory served by these trains are requested to make use of them, inasmuch as their comfort and pleasure have been especially arranged for by having them travel together on these trains."

The Executive Offices have on hand a number of old YEAR BOOKS, 1923-24. While these have no present value from the standpoint of directory purposes (the new 1927-28 Year Book was issued during the summer), they may be of interest and historical value to some of our members of more recent election. The Executive Secretary will gladly send a copy to any member upon receipt of 50c in stamps to cover cost of handling and mailing.